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THE INTERPRETATION OF MACLEAN'S UREA CONCENTRATION TEST IN ASSESSING RENAL FUNCTION IN PREGNANCY.

By VERA I. KRIEGER, M.Sc. (Melbourne).

(From the Biochemistry Department, University of Melbourne, and the Women's Hospital, Melbourne.)

PART II.

INDICATIONS OF RENAL INEFFICIENCY IN PREGNANCY WHEN THE PERCENTAGE OF UREA, VOLUME OF URINE AND TOTAL QUANTITY OF UREA EXCRETED ARE TAKEN INTO CONSIDERATION IN MACLEAN'S UREA CONCENTRATION TEST.

In Part I it has been established that when normal concentrating power of the kidney is found in association with a small secretion of urine after fifteen grammes of urea have been given, the total amount of urea excreted in the three hours of the test gives a more satisfactory measure of renal efficiency than the percentage concentration of urea in any of the single hourly specimens in the MacLean urea concentration test. It seemed necessary, therefore, to determine whether in all combinations of concentrating power and volume of urine secreted this total excretion of urea gave a true indication of renal efficiency.

The possible combinations of total urea excreted in three hours in relation to volume of urine secreted are as follows:

1. Normal total urea excreted in a normal volume of urine in three hours.

2. Normal total urea excreted in a small volume of urine in three hours.

3. Normal total urea excreted in a large volume of urine in three hours.

4. Small total urea excreted in a normal volume of urine in three hours.

5. Small total urea excreted in a small volume of urine in three hours.

6. Small total urea excreted in a large volume of urine in three hours.

An analysis was made of the 703 urea concentration tests from 437 pregnant patients already mentioned in Part I, A. In Table I is set out the proportion of tests falling into the groups already defined in each of the types of toxæmia studied, as well as in normal patients. Tests in the pre-delivery period are separated from those taken during the puerperium. The standard of normality used for this classification was that at least four grammes of urea must be secreted in 150 to 300 cubic centimetres of urine during the three hours following ingestion of 15 grammes of urea in 100 cubic centimetres of water.

In Group I, in which a normal total excretion of urea occurs in a normal volume of urine, the majority of tests reveal normal concentrating power. In about 13% of the tests in this group the percentage of urea is somewhat low and the group will therefore include "borderline" cases as well as those in which the kidney is functioning efficiently, for example, a first-hour specimen 1·3% urea in 78 cubic centimetres of urine, second-hour specimen 2·2% urea in 83 cubic centimetres of urine, third-hour specimen 1·7% urea in 84 cubic centimetres of urine, 4·3 grammes of urea being excreted in 245 cubic centimetres of urine in three hours. It is therefore necessary in this group to consider the percentage of urea in each specimen as well as the total excretion of urea, so as to pick out tests indicating mild renal inefficiency. On reference to Table I it will be noted that in the *ante partum* series 60% to 70% of the tests in patients classified as having a normal pregnancy and a normal pregnancy following toxæmic pregnancy are in Group I; 37% to 55% of the tests in mild toxæmias (albuminuria up to four days' duration, mild recurrent toxæmia and pyelitis), and less than 35% of the more marked toxæmias (prolonged albuminuria, preeclampsia, eclampsia, chronic nephritis and severe recurrent toxæmia) are also in Group I.

Either concentrating power or total excretion of urea in three hours may be used as a measure of renal function in Group II when normal total urea is excreted in a small volume of urine in three hours. If an antidiuretic principle is active in pregnancy and more marked in toxæmic pregnancy, the percentage urea may indicate a degree of renal efficiency greater than that actually existing. This effect will be produced in any condition associated with retention of water in blood and tissues. The three hours' excretion of urea would thus give a more accurate idea of actual renal efficiency.

Two types of urea concentration test are included in Group III. In the first group (IIIa) a normal concentration of urea is found with a large volume of urine, and hence a large total quantity of urea

Table I.

A Comparison of the Percentage Tests in Groups I to VI in the various Toxæmias of Pregnancy and in Normal Pregnant Patients.

Clinical Groups.	*Ante Partum.* Number of Cases.	*Ante Partum.* Number of Tests.	*Ante Partum.* Percentage Tests in Group I.	II.	IIIa.	IIIb.	IV.	Va.	Vb.	VI.	*Post Partum.* Number of Cases.	*Post Partum.* Number of Tests.	*Post Partum.* Percentage Tests in Group I.	II.	IIIa.	IIIb.	IV.	Va.	Vb.	VI.
Normal	63	70	64	7	4	6	7	9	1	1	13	14	43	7	29	14		7		
Normal following toxæmic pregnancy	26	34	68		3	6	6	14		6	20	20	70		20	5				
Albuminuria of labour	40	43	37	12	2	9	16	7	12	4	37	37	51	5	22	11	5			
Albuminuria of up to four days' duration	47	60	33	12	7	3	23	15	8	10	8	10	40	20	10	5	11			
Pyelitis	90	116	16	12	1	8	15	25	13	12	25	91	48		11	21	30	2	6	3
Albuminuria of more than four days' duration	2	3							100		5	6	17		17	17	9		17	34
Preeclampsia	11	15	20			7	14	7	27	27	24	34	35		14	18	14			18
Eclampsia																				
Recurrent toxæmia—																				
Mild	23	29	55	7	10	7	3	7	10	4	13	14	57		7	29	7		3	21
Severe	26	32	57	4	2		39	13	13	11	26	38	40		4	9	5			12
Chronic nephritis	8	9	22				44	11	11		8	8	50			12	25			
Total	336	431									239	272								

703 tests on 437 patients. Some of the cases *ante partum* are repeated in *post partum* group.

is excreted. In such tests the renal efficiency is normal. The second type of result is characterized by low concentration of urea excreted in a large volume of urine, with a resultant normal urea excretion in three hours. It is necessary here to consider tests in the *ante partum* and *post partum* periods separately, since different factors may be responsible for the volume of urine secreted. The increased secretion of urine is noted in relatively few *ante partum* tests (Table II), except in the eclamptic group. The figures in this class are probably not representative, since in few cases can tests be made until after delivery. It has been well established that oliguria is characteristic of eclampsia.

The relatively few instances of polyuria in these cases in which *ante partum* tests were made, in spite of the fact that toxæmic patients are instructed to drink a large quantity of fluid per day, indicates the possibility of some antidiuretic effect. Patients in whom polyuria does occur may therefore be free from antidiuretic influence, and because of the large fluid intake will secrete a large volume of urine with possibly lowered urea concentration. On the other hand, the polyuria may simply be due to the compensatory activity of a damaged kidney eliminating waste products to the normal extent by excreting a large volume of dilute urine. The interpretation of tests in this group is therefore uncertain, if either the concentrating power or the excretion of urea in three hours is used as the measure of efficiency. In the puerperium, urea concentration tests show a much higher percentage with increased secretion of urine. In the more severe toxæmias 50% of the tests reveal volumes in excess of 300 cubic centimetres for three hours (Table II). This diuresis may be due to several factors. Usually there is ingestion of large volumes of fluid from four to eight days *post partum* (eight pints per day). Frequently, when very small secretions of urine are noted before delivery, the *post partum* test is characterized by a large volume of urine. If there is an antidiuretic hormone which is no longer present in the blood after delivery, the increased fluid intake at this time without the opposing antidiuretic action would satisfactorily explain these results. If the generally accepted idea of a "toxin" as the cause of the toxæmia is held, removal of the toxic factor after delivery, resulting in the release of œdema fluid, might afford an explanation of the volume observed. Finally the compensatory effect of the damaged kidney cannot be overlooked. Hence the interpretation of urea concentration tests in which a low percentage of urea is excreted in a large volume of urine resulting in normal total excretion of urea, is uncertain. The low concentrating power may be due to release of accumulated fluid from the tissues, to ingestion of large volumes of fluid or to the inefficiency of a severely damaged kidney. Tests in such cases should be repeated after the patient has had restriction of fluid for a longer period than that specified earlier in the paper.

TABLE II.

A Comparison of the Percentage of Urea Concentration Tests in which over 300 Cubic Centimetres of Urine were Excreted in Three Hours following Ingestion of 15 Grammes of Urea by Normal and Toxæmic Pregnant Women.

Type of Patient.	*Ante Partum.*	*Post Partum.*
	%	%
Normal pregnant	14	40
Normal pregnant following toxæmic pregnancy	17	—
Albuminuria of labour	—	21
Albuminuria of four days' duration ..	16	27
Albuminuria of more than four days' duration	21	45
Eclampsia	31	50
Preeclampsia	—	50
Chronic nephritis	14	22
Recurrent toxæmia	15	48
Pyelitis	10	30

In Group IV less than four grammes of urea is excreted in three hours in a normal volume of urine. This division mainly includes tests interpreted as indicating poor renal function, since in 85% the concentrating power is also low. A few tests (15%) are included in which a high concentration of urea is found in one specimen only and then in a small volume of urine whilst the other specimens show low concentrating power, the total excretion being low. The normal concentrating power in one specimen would be interpreted by MacLean's method as indicating normal renal function; for example, the first-hour specimen 1·7% urea in 89 cubic centimetres of urine, the second-hour specimen 1·4% urea in 75 cubic centimetres of urine, the third-hour specimen 2·5% urea in 12 cubic centimetres of urine. The total excretion of urea was three grammes in 176 cubic centimetres of urine in three hours. This test was made in a case of severe recurrent toxæmia of pregnancy.

In the previous pregnancy the patient had been classed as a chronic nephritic. Throughout this earlier pregnancy there had been pronounced persistent albuminuria and persistent high blood pressure as well as optic neuritis and albuminuric retinitis. The test quoted was made when the patient was about four months pregnant, and later the results of the renal function tests were very poor.

In this case the total excretion of urea was a more accurate measure of the renal efficiency than the concentrating power. The majority of tests of this type in Group IV were made in cases of persistent albuminuria, recurrent toxæmia of pregnancy and pyelitis.

In this group, therefore, the total excretion of urea in three hours is of definite value in indicating renal damage not always assessed by the concentrating power. In Table III is set out the distribution of the types of results of the test in Group IV, and it is significant that in 92 cases only six tests of this type occur in normal pregnant women, normal pregnant women following a toxæmic pregnancy, and patients with albumin of labour.

Tests in this group indicate renal damage ranging from very mild derangements to marked involvement.

In urea concentration tests in which a small amount of urea is excreted in a small volume of urine in three hours, two types of test must again

TABLE III.

A Tabulation of Tests revealing a Small Excretion of Urea in Three Hours secreted in Normal Volume of Urine. (Group IV.)

Type of Patient.	Total Tests revealing Small Excretion of Urea in Normal Volume of Urine in Three Hours.	Number of Tests revealing Concentrating Power Normal in one Specimen only, but Total Excretion in Normal Volume of Urine Low.
Normal pregnant	4	1
Normal pregnant following toxæmic pregnancy	2	—
Albuminuria of labour	—	—
Albuminuria up to four days' duration	11	1
Albuminuria of more than four days' duration	24	3
Preeclampsia	—	—
Eclampsia	7	1
Chronic nephritis	5	—
Recurrent toxæmia	22	3
Pyelitis	17	5
Total	92	14

be considered. In Group V*a* the concentrating power is high or normal, but the urea is excreted in only a small volume of urine, hence the total excretion of urea in three hours is less than four grammes. This combination has been discussed in detail in Part I, A, and it has been proved that the concentrating power does not represent the true renal function under the specified conditions. When the concentrating power is low and the secretion of urine is small, as in Group V*b*, there is no doubt as to the renal impairment. Here the kidney is unable to concentrate urea to the normal degree, in spite of a very small secretion of urine. Only one test of this type was found in patients in the normal pregnant group, normal pregnancy following toxæmic pregnancy and among patients with albuminuria of labour, whilst the greatest number were found among the severely toxæmic patients. There is no doubt that this type of test indicates severe renal damage.

In Group VI a small concentrating power is combined with a large excretion of urine, but the amount of urea excreted in three hours is less than four grammes. Thus in spite of the attempted compensation by increased secretion of urine, the kidney is unable to excrete the normal amount of urea. This combination indicates, therefore, a definite renal involvement. Few tests of this type are obtained in the normal conditions of pregnancy, but many are obtained in the severe or prolonged toxæmias. This is particularly noticeable in tests made during the puerperium following toxæmic pregnancy.

Comment on the Clinical Data of the Cases Investigated.

The incidence of urea concentration tests in the groups already described is summarized in Table I. The clinical data available for the patients in this series of investigations and correlated with the urea concentration test interpreted in terms of the total urea excreted in three hours following ingestion of 15 grammes of urea, are as follows. Among the 70 patients in the normal pregnant group 12 tests indicated renal inefficiency and the previous history of the patient was in agreement with the possibility of such inefficiency. In the series of 26 patients with a normal pregnancy following a toxæmic pregnancy and who now gave normal results to urea concentration tests, there were nine cases in which, because of previous severe toxæmia, there was a possibility of renal damage. No evidence of renal inefficiency was present in this pregnancy.

Among 20 patients with albumin of labour normal results were obtained to tests in the puerperium in all cases except one in which there had been persistent albuminuria, high blood pressure and œdema in an earlier pregnancy.

When albumin persisted up to four days 43 tests were made on 40 patients in the *ante partum* period, and 17 tests (40%) indicated renal inefficiency. From the clinical point of view there were 11 tests on patients with mild albuminuria and high blood pressure, and four on patients with marked albuminuria. Œdema, headache and eye signs were noted among these patients; two were delivered of premature babies and in one case labour was induced.

In the puerperium there were 37 tests on 37 patients, and only four tests revealed renal inefficiency. Two of these patients had considerable albuminuria with œdema, and the third had a history of nephritis and tonsillitis.

In the group of prolonged albuminurias there were 73 tests out of 116 (63%) indicating renal inefficiency, and a further eight tests occurred in Group III*b*, in which the interpretation is uncertain. Among the patients who gave such results to the tests, persistent albuminuria up to one month was noted, in two patients labour was induced and one patient was delivered of a stillborn baby.

Of the 16 patients whose tests occurred in Group IV, 11 had very high blood pressure, 6 had marked œdema, and 10 had had a previous severely toxæmic illness, such as scarlet fever or tonsillitis. In one case there was a macerated baby, and in all cases albuminuria was persistent. Among the 24 patients whose tests fell in Group V*a*, were 13 who had severe persistent albuminuria and high blood pressure. There were six premature babies, four stillbirths, one macerated stillborn baby, and two patients had labour induced. Œdema, headache, eye signs and oliguria were common among these patients. Twelve patients had tests in Group V*b*. Six of these had mild but persistent albuminuria, and six were severe albuminurics with high blood pressure. There were six premature births (four being stillbirths), one macerated fœtus and six inductions of labour. In Group VI there were eight patients with persistent albuminuria and high blood pressure. Labour was induced in three cases, there were two premature babies and one stillborn macerated babe.

In the puerperium 59% of the urea concentration tests gave normal results. Only 21% of these had been normal before delivery. A further 9% had

previously been in Group III*b*, in which the interpretation is doubtful. Twenty *per centum* of the patients gave positive results to tests in Groups IV, V*a*, V*b* and VI in the puerperium. Eight of these had severe persistent albuminuria and high blood pressure before delivery. There were five inductions of labour, three premature births, three stillbirths, and one patient was delivered of a macerated fœtus. In seven cases pre-delivery tests had indicated renal inefficiency, and in three the *ante partum* tests gave normal results. In one case the blood urea had been 58 milligrammes per 100 cubic centimetres of blood in the *ante partum* period.

In preeclampsia there were only three tests before delivery, and all were in Group V*b*. These patients all had severe albuminuria and high blood pressure, and in two labour was induced. These latter had a history of scarlet fever. Four tests were made in the puerperium; two of these patients had severe toxæmia and had labour induced, and *post partum* tests gave normal results. In the other two cases *post partum* tests fell in Group VI.

Eight of the fifteen eclamptic patients tested before delivery had urea concentration tests in Groups IV, V*a*, V*b* and VI. These patients each had severe albuminuria and high blood pressure, two had œdema, and three eye symptoms. In five cases labour was induced and the number of fits varied between one and nine. Thirty-four tests were performed on 24 eclamptic patients in the puerperium, and 17 of these tests gave normal results. Six tests fell in Group III*b*, and in these cases the clinical data left no doubt as to the renal involvement. In Groups IV and VI there were 11 tests, and the patients suffered severe eclamptic attacks characterized by marked albumin, high blood pressure, blurred vision, headache, œdema and fits.

Among those patients diagnosed as chronic nephritics, seven of the nine tests on the eight patients were in Groups IV, V*a*, V*b* or VI. A characteristic feature in these cases was the fact that tests revealing poor function were obtained early in pregnancy, in one case when the patient was only three months pregnant. In two cases the result of the Fowweather clearance test was low, and in one the blood urea was 218 milligrammes. Labour was induced in three cases. In the puerperium four of the eight tests gave normal results in spite of the marked toxæmia during the pregnancy. In one case the blood urea was 81 milligrammes. One patient had a history of nephritis since she was a child; her clearance according to the Fowweather test was only 11%. Her labour was induced. Another patient, who had a *post partum* test made in Group VI and who collapsed in the street, had albuminuric retinitis and her baby was stillborn.

The recurrent toxæmias of pregnancy were divided into mild and severe groups. In the mild recurrent group were fourteen patients whose renal function tests now gave normal results, and these included ten albuminurics; three of them had labour induced in the earlier pregnancy, three eclamptics and one cardiac patient in the previous pregnancy. Two patients previously albuminuric now had tests made in Group III*b*. In Groups IV, V*a* and V*b* were six patients with severe toxæmia in a previous pregnancy, and two of these had had labour induced.

Among the severely toxæmic patients in the present pregnancy were 17 whose tests now gave normal results, four in Group III*b* (uncertain) and 31 whose tests definitely indicated renal inefficiency. In Group III*b* one patient had had three toxæmic pregnancies; she had induction of labour in one of them and was severely toxæmic in the present pregnancy.

In Group IV there were 11 patients with recurrent toxæmia, and only two of these had mild albuminuria in the present pregnancy. Two had previously had severe albuminuria; this now recurred, and in both patients labour was induced. A patient, previously preeclamptic, at two months had albuminuria and a systolic blood pressure of 180 millimetres of mercury as well as eye symptoms. The systolic blood pressure rose to 200 and the pregnancy resulted in a miscarriage. One patient with severe albuminuria and an eclamptic patient suffered from severe albuminuria in their second pregnancies, and in each case and in each pregnancy Cæsarean section was performed. In four cases severe albuminuria had occurred in two previous pregnancies and again occurred in the present pregnancy. Two of these patients had had inductions in each of the previous pregnancies, and induction was again performed in the third pregnancy. In one of these the blood urea was 104 milligrammes and the result of the Fowweather clearance test was 15% at two months. In the second case the blood urea was 53 milligrammes.

In Group V*a* there were two recurrent albuminurics and in one of these the systolic blood pressure rose to 230 millimetres of mercury and the patient had labour induced. The other patient had had three severe albuminuric pregnancies, the last two resulting in stillbirths. In Group V*b* there were five cases of previous severe albuminuria; in three cases there had been induction of labour and in two Cæsarean section. One patient had had four previous albuminuric pregnancies; in the first labour had been induced and in the present pregnancy "albumin was solid" and the clearance according to the Fowweather test was 15% at eighteen weeks.

Three patients had previously been eclamptics. In one the blood pressure had begun to rise and there was albumin in the urine at fourteen weeks in the present pregnancy. The clinical condition showed no improvement with treatment and the pregnancy was terminated by Cæsarean section. In each of the other patients albumin appeared about a month before delivery and failed to respond to treatment. Labour was induced in one patient.

There was one patient in Group VI who had had severe albuminuria and induction of labour in a previous pregnancy.

Even in patients with severe recurrent toxæmia in which induction of labour had been necessary, tests in the puerperium frequently gave normal results. Many patients, however, did show renal impairment in the puerperium. In Group IV three such cases occurred. In two patients this was the third toxæmic pregnancy. In one case blood urea was 104 milligrammes before delivery and 178 milligrammes *post partum*, whilst the urea concentration was in Group IV in both instances. In another patient there was high systolic blood pressure (210) and albumin persisted throughout the puerperium, and tests in this period were in Groups IV and VI. A third patient, previously eclamptic, had five tests in the pre-delivery period in the present pregnancy, and all fell in Group IV or V*b*.

Two patients with very severe toxæmia had tests in Group V*b* in the present pregnancy. One was previously eclamptic and her systolic and diastolic blood pressure rose to 210 and 140 millimetres of mercury in the present pregnancy. Another patient had suffered two previous severe albuminurias, in one of which labour was induced. Two fairly normal pregnancies followed, and in the present pregnancy albumin in the urine was "solid" for ten days at eighteen weeks. Tests *ante partum* were in Group V*a* and, following hysterectomy, two tests were in Group V*b*. In Group VI there was one case of very severe toxæmia with œdema and very high systolic blood pressure (250). Previously this patient had had three toxæmic pregnancies, and in two induction of labour had been necessary.

In pyelitis of pregnancy 56% of the tests fell in groups indicating renal inefficiency. Fourteen tests occurred in Group IV, and six of the patients had severe toxæmia, two having to have induction of labour, and the result of the Fowweather test was low in three of them. In Group V*b* there were three patients, all with severe toxæmia. In Group VI there was one severely toxæmic patient, who was delivered of a stillborn baby.

Discussion.

The conclusions resulting from this analysis of the possible variations of urea concentration and volume of urine in the urea concentration test in relation to the excretion of urea during the period of the test may be briefly summarized as in Table IV.

It is evident that if the total excretion of urea is used as a measure of renal efficiency, tests in Group I*b* will be interpreted as normal, whilst actually there is some slight degree of inefficiency. In this instance more information is gained from the concentrating power than from the total excretion of urea. In Group III*b*, although the total excretion of urea is normal, the condition of the kidney cannot be assessed with any degree of certainty. In this group the low concentration of urea may be due to a large excretion of urine because of excessive fluid intake, to absence of the influence of an antidiuretic hormone, to release of œdema fluid, particularly in the puerperium, or to the compensatory action of a badly damaged kidney.

From the clinical data it is evident that patients with tests in this group frequently exhibit severe kidney damage.

In this series of 703 urea concentration tests, 241 showed low concentration of urea, and by MacLean's method these would be interpreted as indicating renal inefficiency. Such tests occurred in Groups III*b*, IV, V and VI in this classification. In 128 of these tests the interpretation was certain, since small or normal volumes of urine were secreted and the total excretion of urea was low. Such tests occurred in Groups IV*a* and V*b*. In 113 tests low concentration of urea was excreted in large volumes of urine, and this type of test was divisible into two subgroups: (i) a group in which the total excretion of urea was normal, Group III*b*, (ii) a group in which the total excretion of urea was low, Group VI.

In Group VI the kidney is unable to excrete the normal amount of urea in spite of secretion of large volumes of urine.

From a consideration of the clinical data presented it is evident that there is agreement between the clinical condition of the patient and the renal efficiency assessed by the total excretion of urea in the MacLean urea concentration test. From Table III it is evident that over 50% of the tests on patients in severe and prolonged toxæmias exhibited renal inefficiency in the *ante partum* period, that is, tests occurred in Groups IV, V and VI. In the puerperium a greater percentage of patients with poor renal function were again found among the toxæmic patients.

Table IV.

The Relation of Concentrating Power, Total Excretion of Urine in Three Hours, and the Total Excretion of Urea in Three Hours, to Renal Efficiency.

Group.	Concentrating Power.	Total Volume of Urine in Three Hours.	Total Excretion of Urea in Three Hours.	Kidney Function.
I. Normal urea excretion in normal volume in three hours	(*a*) Normal.	Normal.	Normal.	Normal.
	(*b*) Slightly low.	Normal.	Normal.	Mild inefficiency.
II. Normal urea excretion in small volume in three hours..	Normal.	Small.	Normal.	Normal.
III. Normal urea excretion in large volume in three hours..	(*a*) Normal.	Large.	Normal.	Normal.
	(*b*) Low.	Large.	Normal.	Uncertain.
IV. Small urea excretion in normal volume in three hours..	(*a*) Low.	Normal.	Small.	Mild ——→ Severe Inefficiency.
	(*b*) One value normal in small volume, two values low.	Normal.	Small.	
V. Small urea excretion in small volume in three hours ..	(*a*) Normal.	Small.	Small.	Renal inefficiency.
	(*b*) Low.	Small.	Small.	Severe renal damage.
VI. Small urea excretion in large volume in three hours ..	Low.	Large.	Small.	Renal inefficiency.

TABLE V.

A Comparison of the Percentage of Cases with Renal Function Tests in Groups IV, V and VI in Normal and Toxæmic Pregnancy, and in the Puerperium.

Type of Patient.	*Ante Partum.*		*Post Partum.*	
	Total Number of Tests.	Percentage Tests in Groups IV, V, VI.	Total Number of Tests.	Percentage Tests in Groups IV, V, VI.
Normal pregnancy	70	18	14	7
Normal pregnancy following toxæmic pregnancy	34	26	—	—
Albuminuria of labour	—	—	20	5
Albuminuria up to four days	43	39	37	11
Albuminuria of more than four days' duration	116	65	91	20
Preeclampsia	3	100	6	51
Eclampsia	15	75	34	32
Recurrent toxæmias—				
Mild	29	20	14	—
Severe	52	59	38	10
Chronic nephritis	8	77	8	37
Pyelitis	60	56	10	30

Tests in Group VI may therefore be interpreted as definitely indicating renal inefficiency. Forty-five tests of this type occurred in the series, and the separation of this type of test with low total excretion of urea from those with normal total excretion in large volume (Group III*b*) reduces the number of tests where interpretation was "uncertain" from 16% to 9%.

In Group IV a small number of tests were found in which normal urea concentration was associated with small volume of urine in one specimen, while low urea concentrations were obtained in the other two specimens. This combination resulted in low total excretion of urea, and these tests were found to occur among cases clinically representing renal impairment. Fourteen tests of this type were observed.

Because of normal concentrating power, tests in Group V*a* would ordinarily be classified as normal renal function, but owing to the small volume of urine secreted, the total excretion of urea is less than four grammes per three hours. In such cases it has been established (Part I, A) that renal inefficiency exists. There were 65 such tests in the series investigated.

The total number of tests in which the total excretion of urea per three hours aids in determining kidney function is thus (i) 79 previously termed normal and shown to represent inefficiency (Groups IV*b* and V*a*), (ii) 45 where the efficiency was "uncertain" and have been definitely established as "inefficient" (Group VI). By modifying the interpretation of the MacLean urea concentration test so that efficiency is determined by total excretion of urea, 17·6% more tests were shown to indicate renal inefficiency than when concentrating power was used as the determining factor. For comparison an analysis of urea concentration tests reported by Cruickshank, Hewitt and Couper into the six groups described in the present paper is summarized in Table VI.

If Groups I, II and III*a* be termed normal renal function, Group III*b* uncertain function, and Groups IV*a* and IV*b*, V*a*, V*b* and VI renal inefficiency, it will be observed (Table VII) that among the normal pregnant patients 69% of tests fell in the normal groups and 20% in the groups indicating renal inefficiency. In the severe toxæmias (preeclampsia, nephritis and eclampsia) only one-third to one-fifth of the tests showed normal renal function and one-half to two-thirds indicated renal damage. Among the less severe toxæmias (albumin-

TABLE VII.

Percentage of Tests in the series by Cruickshank, Hewitt and Couper occurring in groups indicating Normal Renal Function, Uncertain Renal Function, and Renal Inefficiency.

Clinical Types.	Normal Renal Function Percentage Tests.	Uncertain Renal Function Percentage Tests.	Renal Inefficiency Percentage Tests.
Normal pregnant	69	11	20
Albuminuria	40	23	37
Preeclampsia	33	15	52
Nephritis	22	10	68
Eclampsia	34	18	48

TABLE VI.

Distribution of Urea Concentration Tests on 220 Normal and Toxæmic Pregnant Women in a series reported by Cruickshank, Hewitt and Couper into the Six Groups defined earlier.

Clinical Types.	Number of Tests.	Group I.	Group II.	Group III*a*.	Group III*b*.	Group IV*a*.	Group IV*b*.	Group V*a*.	Group V*b*.	Group VI.
Normal pregnant	39	22	—	5	4	1	—	5	1	1
Albuminuria	51	17	1	2	12	8	—	4	4	3
Preeclampsia	33	8	3	—	5	4	—	6	6	1
Nephritis	41	6	—	3	10	8	1	6	9	4
Eclampsia	56	17	1	1	4	6	—	7	11	3
Total	220	70	5	11	35	27	1	28	31	12

uria) the highest incidence of "doubtful" results occurred.

The incidence of tests indicating normal function or renal damage is thus in agreement with the severity of the toxæmia in this series of results obtained under entirely different experimental conditions from those described earlier.

In 105 tests in this series low concentrating power was found with small, normal or large volumes of urine. In 31 of these tests (29·5%) the volume was small, hence the total excretion of urea was low. These tests, either by concentrating power or total excretion of urea, indicated poor renal function. Thirty-five tests (33·5%) fell in Group III*b*, in which the interpretation is uncertain. Twenty-seven tests fell in Group IV*a* and twelve in Group VI. These latter groups indicate renal inefficiency, hence only 33·5% instead of 70·5% of tests were now difficult to interpret.

Twenty-three tests fell in Group V*a* and one in Group IV*b*, in which there is normal concentrating power but low total excretion of urea, and in which clinical evidence indicates renal damage.

Thus in this series of tests 63 tests in 220 (that is, 28·6%) were assessed with greater accuracy. The agreement between the results of the analysis of these tests by Cruickshank, Hewitt and Couper and those found in the present series stresses the importance of this modification of interpretation of the MacLean urea concentration test.

Conclusion.

If, in determining renal function in the toxæmias of pregnancy, the urea concentration test of MacLean is interpreted not only in terms of concentrating power, but by concentrating power in association with volume of urine, that is, by the total excretion of urea during the period of the test, a much more exact idea of renal efficiency is obtained.

Fallacies introduced by assessing renal function by concentrating power alone in conditions in which small volumes of urine are secreted are eliminated, and when there are abnormally large volumes of urine, resulting in low concentrating power, the number of tests in which the interpretation is "uncertain" is lessened. Thus a much closer correlation between chemical test and clinical data is obtained.

Summary.

1. An analysis is made dividing urea concentration tests into groups depending on the relationship of total excretion of urea to small, normal or large secretion of urine.

2. The relationship of these groups to renal efficiency is worked out.

3. A correlation is made between the tests in these groups and the clinical data relating to the patients on whom the tests were performed.

4. The use of these groups in determining renal efficiency is discussed.

5. A comparison is shown between the results of this analysis and one similarly derived from data published by Cruickshank, Hewitt and Couper.

6. Renal function is normal when a normal percentage of urea is excreted in normal or large volume of urine so that there is normal or high total excretion of urea in three hours.

7. Renal damage is indicated: (i) when low percentage of urea is excreted in small volume of urine, resulting in low total excretion of urea in three hours; (ii) when low percentage of urea is excreted in a large volume of urine, but the total excretion of urea in three hours is low; (iii) when normal percentage of urea is excreted in small volume of urine so that the total excretion of urea is low; (iv) when the percentage of urea for one of the three hours is normal or high, but excreted in a small volume of urine, and the percentages of urea in the two remaining samples are low so that the total excretion of urea in three hours is low.

8. When a small percentage of urea is excreted in a large volume of urine so that the total excretion of urea is normal or high, the interpretation is uncertain.

9. The conclusion is drawn that by division of urea concentration tests into these groups a more accurate means of measuring renal efficiency is obtained.

Reference.

(1) J. N. Cruickshank, J. Hewitt and K. L. Couper: "The Toxæmias of Pregnancy: A Clinical and Biochemical Study", Medical Research Council, Special Report Series, 1927-1928, pages 113-120.

THE PROPHYLAXIS OF GAS GANGRENE IN MAN.[1]

By W. J. PENFOLD, M.B., Ch.M. (Edinburgh), D.P.H., B.Hyg. (Durham), M.R.C.S. (England), L.R.C.P. (London),

AND

JEAN C. TOLHURST, M.Sc. (Melbourne).

(From the Baker Medical Research Institute, Alfred Hospital, Melbourne.)

IN a former publication(1) we described the immunization of animals with alum-precipitated formol-toxoid made from *Bacillus welchii* toxin. We demonstrated that two injections of alum-precipitated toxoid, given subcutaneously with an interval of four weeks, protected guinea-pigs against ten minimum lethal doses of living *Bacillus welchii* culture. We also immunized rats and mice, and found that they could withstand several minimum lethal doses of culture. The serum from immunized guinea-pigs was shown to have passive protecting power in mice.

Our experiments in animals had been so successful that in August of last year we decided to use the vaccine in man.

[1] Read at the meeting of the Alfred Hospital Clinical Society on February 22, 1938.

An eighteen-hour culture of *Bacillus welchii* grown in anaerobic veal broth containing 0·1% of glucose was filtered through a Seitz bacterial pad, and the minimum lethal dose of the filtrate was determined intraperitoneally in mice. Only toxins which killed both of two mice in 0·05 cubic centimetre or less were used to prepare toxoids for human subjects. About 0·4% of formalin was added to a freshly prepared toxin and the mixture was incubated at 37° C. for six or seven days, until the toxicity was so reduced that one cubic centimetre would not kill mice of 20 grammes weight, while five cubic centimetres would kill rats of 100 grammes weight.

A sample of toxoid was titrated with an 8% solution of alum to find the amount of alum which gave the maximum precipitate. The bulk of toxoid was precipitated at this point, and after removal of the supernatant fluid the precipitate was washed twice with 0·3% tricresol-saline solution. The sterility of the final suspension was tested aerobically on blood agar and anaerobically in Colebrook's anaerobic meat broth containing active trypsin.

The immunizing power of the material was tested in guinea-pigs.

As we were in doubt as to the severity of the reactions which might result from the injection of alum-precipitated *Bacillus welchii* toxoid in man, the first volunteer received only 0·25 and 0·5 cubic centimetre subcutaneously, with an interval of four weeks between the doses. The reactions were very mild. The site of inoculation was inflamed, slightly swollen and tender to pressure. The area of redness reached a maximum about thirty hours after the injection and was approximately 5·0 centimetres (two inches) in diameter after the first dose, and 7·5 centimetres (three inches) in diameter after the second dose. It subsided rapidly, leaving a small indurated nodule, which disappeared within a few weeks. There was no general reaction.

We have since injected ten different persons with doses ranging from 0·25 to 1·0 cubic centimetre and have encountered no serious reactions.

The immunity response in the first volunteers is most readily demonstrated by a series of charts. The antitoxin was titrated by the intradermal method in guinea-pigs (Glenny *et alii*[2]) and before immunization each person had less than one seventy-fifth of a unit of antitoxin per cubic centimetre of serum. In each chart the time in weeks is shown by the abscissa, and the units of antitoxin by the ordinate. The scale is varied for convenience. The injections are indicated by crosses.

The first chart shows the production of antitoxin in the serum of the first volunteer.

Two weeks after the first small dose of 0·25 cubic centimetre of the toxoid she had one-tenth of a unit of antitoxin per cubic centimetre of serum, while four weeks after the second injection of 0·5 cubic centimetre of toxoid she reached a maximum titre of one-quarter of a unit. The antitoxin fell to one-tenth of a unit, remained at that level for five weeks and fell again to one-twentieth of a unit. Now, six months after the first injection, there is only one-fortieth of a unit of antitoxin per cubic centimetre of serum.

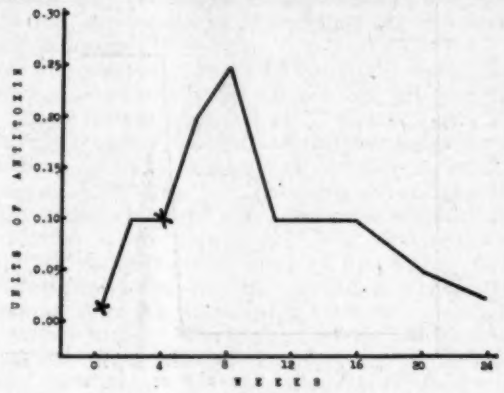

CHART I.
Two injections, 0·25 and 0·5 cubic centimetre.

We determined to try four more volunteers with the same dosage and the same interval. The response was disappointing; in three of the four there was less than one seventy-fifth of a unit of antitoxin per cubic centimetre of serum. We therefore gave them all a third injection of one cubic centimetre of toxoid.

Chart II shows the behaviour of one of these volunteers.

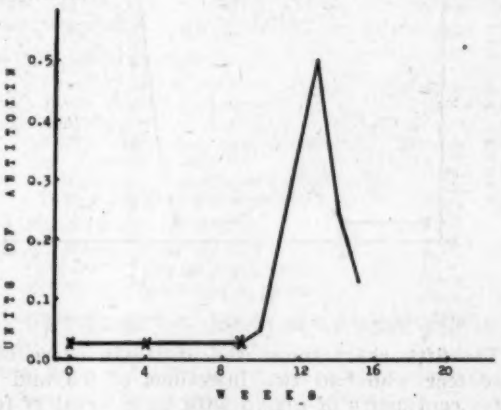

CHART II.
Three injections, 0·25, 0·5 and 1·0 cubic centimetre.

One week after the third injection he had one-twentieth of a unit of antitoxin per cubic centimetre of serum, and the titre rose steadily to a maximum of half a unit three weeks later. Then it fell sharply and is now about one-seventh of a unit.

Chart III represents the antitoxin curve of the third volunteer.

There was no apparent response until after the third injection, but a week later we demonstrated one-sixth of a unit of antitoxin per cubic centimetre of serum. The titre has remained at this level for four weeks.

The fourth chart is interesting because there was some detectable antitoxin after the second injection.

This disappeared rapidly, however. After the third injection there was an increase in antitoxin up to one-fifth of a unit.

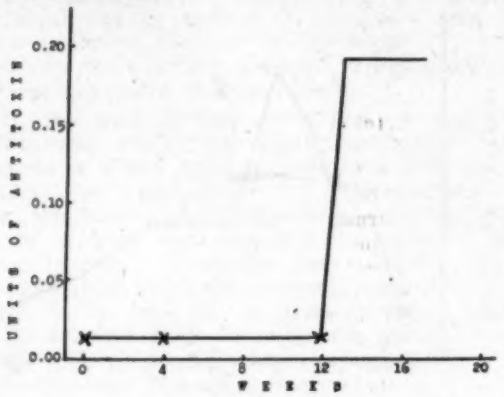

CHART III.
Three injections, 0·25, 0·5 and 1·0 cubic centimetre.

In a fifth volunteer no antitoxin could be demonstrated after the second injection, but two weeks after the third dose his serum contained one-tenth of a unit of antitoxin per cubic centimetre.

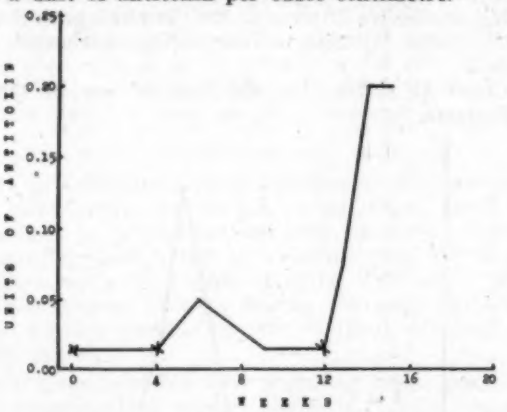

CHART IV.
Three injections, 0·25, 0·5 and 1·0 cubic centimetre.

The fifth chart shows the antitoxin curve of a volunteer who had two injections of 0·5 and 1·0 cubic centimetre of toxoid with an interval of four weeks. In the earlier volunteers the first two doses had been smaller, only 0·25 and 0·5 cubic centimetre. Four weeks after the second dose this person had one-fifth of a unit of antitoxin per cubic centimetre of serum.

The curves in this series vary to some extent, but the fact that stands out is that alum-precipitated *Bacillus welchii* toxoid given in two or three injections stimulated the production of antitoxin in all of six persons.

There are five other volunteers whose course of immunization is not yet complete; hence there are no results available for them.

In order to demonstrate that the immune human serum was fully protective against living *Bacillus welchii* culture, we carried out passive immunity experiments in mice. Table I shows the results.

TABLE I.
Passive Immunity in Mice.

Group.	Dose of Culture in Cubic Centimetres.	Fraction Survived.
(1) Immune human serum	0·20	4/4
(2) Normal human serum	0·20	0/4
(3) No serum	0·20	0/4
(4) No serum	0·15	0/2
(5) No serum	0·10	0/2

0/12

We gave four mice each about one unit of antitoxin from the second immunized volunteer, by injecting two doses of one cubic centimetre of serum intraperitoneally with an interval of six hours. Four control mice each received the same volume (2·0 cubic centimetres) of normal human serum. The next day all eight mice were given 0·2 cubic centimetre of *Bacillus welchii* culture intraperitoneally. As shown by the table, the four mice which had received immune serum were protected and all survived, while those with normal serum and those with no serum all died. The fact that 0·1 cubic centimetre of culture killed both of two mice means that the protected mice withstood at least two minimum lethal doses of culture.

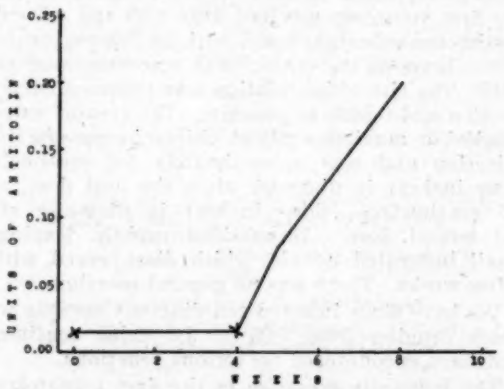

CHART V.
Two injections, 0·5 and 1·0 cubic centimetre.

Throughout this work we have used a strain of *Bacillus welchii*, Type A, obtained from the Mulford Laboratories in Pennsylvania. Serum from rabbits immunized with toxoid and toxin made from this strain has been found to be fully protective against five foreign strains and six local strains of *Bacillus welchii*. The local strains were all isolated from patients at the Women's Hospital, Melbourne, during the last six months.

The evidence presented here suggests that we now have a suitable material for immunizing man against gas gangrene due to *Bacillus welchii*, although we cannot at the moment determine what level of antitoxin is fully protective. We are still not quite satisfied as to the best method of

immunization with regard to the size of the doses and the intervals between them, nor are we yet in a position to discuss the duration of immunity in man. We know that in the first volunteer (Chart I) antitoxin could be detected for a period of nearly six months. At a later date we propose to determine the effect in this person of another injection of toxoid.

The indications for the use of our prophylactic are discussed in our first paper.

Recent work abroad[3] has shown that a mixture of tetanus toxoid and diphtheria toxoid precipitated with alum makes an effective immunizing agent against both diseases. We have reason to hope that the alum precipitate of a mixture of tetanus and *Bacillus welchii* toxoids would be equally useful.

Acknowledgements.

The work of one of us (J.C.T.) has been continued with the financial support of both Mr. W. M. Hughes, as Minister of Health of the Commonwealth, and the trustees of the Returned Sailors and Soldiers' Children's Scholarship Fund of Victoria, to which authorities we are deeply indebted.

Our thanks are due to Miss H. M. Butler for the strains of *Bacillus welchii* obtained from the Women's Hospital, Melbourne.

We are indebted to the volunteers, chiefly laboratory colleagues, who have made this work possible, and also to Mr. Douglas Wilson for technical assistance.

References.

(1) W. J. Penfold and Jean C. Tolhurst: "Formol-Toxoids in the Prophylaxis of Gas Gangrene", THE MEDICAL JOURNAL OF AUSTRALIA, June 26, 1937, page 982.
(2) A. T. Glenny, Mona Llewellyn-Jones and J. H. Mason: "The Intracutaneous Method of Testing the Toxins and Antitoxins of the 'Gas Gangrene' Organisms", *The Journal of Pathology and Bacteriology*, Volume XXXIV, 1931, page 201.
(3) F. G. Jones and James M. Moss: "The Antitoxic Titers of Human Subjects following Immunization with Combined Diphtheria and Tetanus Toxoids, Alum Precipitated", *Journal of Immunology*, Volume XXXIII, 1937, page 173.

LUPUS VULGARIS AND THE SIGNIFICANCE OF CERTAIN NON-SPECIFIC ERUPTIONS IN RELATIONSHIP TO TUBERCULOSIS.[1]

By J. E. McGLASHAN, M.C., M.B., B.S.,
Perth.

For more than seventy years the manifestations of cutaneous tuberculosis have been generally recognized. Some forms, however, have been the subject of controversy, and the situation even today is far from clear. Classifications have been made on (*a*) bacteriology and animal inoculation, (*b*) histology and microscopic structure, (*c*) ætiology. Until these different methods give identical results there must be a difference of opinion, and each man will judge as his experience suggests to him.

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

There have been one or two notable rearrangements—the rosacea-like tuberculide of Lewandowsky has been separated from rosacea into its proper grouping, and *granulosis rubra nasi* has been put with the syndrome of acrodynia of childhood (Swift's disease).[1]

For the present it must be said that cutaneous tuberculosis can only be classed as (*a*) proved, (*b*) approved, (*c*) disputed.

Darier[2] has laid down certain postulates for a cutaneous lesion to be regarded as tuberculous, the principal ones being: (i) the presence of tubercle bacilli in its structure; (ii) the positive result of inoculation in the guinea-pig; (iii) focal reaction in the lesion itself on injection of tuberculin. The next characteristics authorize a sufficiently strong presumption of the lesion being tuberculous only if several are united. They are: (iv) the histological structure, which must conform to that of lesions known certainly to be tuberculous; (v) a certain clinical aspect—evolution and special ætiological circumstances; (vi) coexistence with lesions indubitably tuberculous. The types conforming to all these postulates are remarkably few. They are: (*a*) tuberculous ulcer, (*b*) *tuberculosis cutis verrucosa*, (*c*) scrofulodermia (*tuberculosis colliquativa*), (*d*) fungoid tuberculosis and (*e*) *lupus vulgaris*. These Darier refers to as "tuberculoses". We can call this Group I.

Our second group does not conform to the main postulates (i) and (ii), but it does conform to the others. This group Darier labels "tuberculides". These tuberculides are classified as follows:

(*a*) Cutaneous tuberculides:
- *Tuberculosis lichenoides* (*lichen scrofulosorum*).
- *Tuberculosis papulonecrotica*.
- Acneiform tuberculosis.
- *Tuberculosis lupoides* (sarcoid of Besnier and sarcoid of Boeck).
- Atrophiant erythematous tuberculosis.
- Diffuse *lupus erythematosus*.

(*b*) Hypodermic tuberculides:
- *Erythema induratum*.
- Sarcoid of Darier and Roussy.
- Nodular disseminated sarcoid.

Darier regards the three last as being probably similar.

Time does not permit my detailing other classifications, such as those of Lewandowsky, of Gans, of Montgomery *et cetera*, and I shall proceed by mentioning the dermatoses in the order suggested by Funk, who has classified the tuberculous lesions according to the degree of immunity of the patient's organism:

Class I: Defective immunity—
- (*a*) Miliary tuberculosis of the skin.
- (*b*) Ulcerous miliary tuberculosis or tuberculous ulcer (orificial).

This group need not detain us.

Class II: Moderate immunity of the organism—
- (*a*) *Lupus vulgaris*.
- (*b*) Disseminated miliary or follicular lupus.
- (*c*) *Tuberculosis verrucosa cutis*.
- (*d*) *Tuberculosis colliquativa*.

(e) *Tuberculosis serpiginosa, tuberculosis fungosa cutis.*
(f) *Tuberculosis cutis lichenoides.*
(g) *Tuberculosis papulonecrotica.*
(h) *Erythema induratum.*

Classes III and IV consist of lesions of uncertain and disputed origin and may be dealt with later.

It is evident that climatic influences have a pronounced bearing on the incidence and intensity of *lupus vulgaris*. What represents a major social problem in Great Britain and Europe is much less important in the United States of America and is an infrequent event in this country. My colleague, Dr. O'Donnell, and I have the impression that cutaneous tuberculosis is relatively more common in western than in eastern Australia. In recent years I have seen five native-born patients with *lupus vulgaris*, and I have at present a patient who, though English born, lived for thirty years in Western Australia before lupus supervened on a lacerated wound of the forehead. The situation of *lupus vulgaris* near the body orifices and on sites liable to injury, such hands, ears and buttocks, suggests that the infection has been from inoculation, possibly autoinoculation, from sputum *et cetera*. The appearance of multiple foci of lupus occurring simultaneously after measles, however, indicates the probability of the hæmatogenous route.

Before I leave *lupus vulgaris*, perhaps a word or two on treatment may be permitted. Excision, when cosmetic circumstances permit, is still the method of choice. The electric cutting knife is a modern improvement. Excision of the underlying fat and the avoidance of tension on the scar are important factors. The Finsen light treatment still holds its position, but most of you know better than I its expensive equipment, the need of constant skilled attention during treatment, and the time consumed. *Grenz* ray therapy is being largely used in Germany. The digestive method has also been favourably reported on.[3]

Not being able to obtain the compound used in Germany, which contains trypsin, I have been using at weekly intervals pepsin under "Elastoplast", and the result has been astonishing. I am not altogether sure that "Elastoplast" has not been the main factor, and I believe that at least for the preliminary treatment of *lupus vulgaris excedens* there is here a valuable remedy. The salt-free diet of the type of Gerson, Sauerbruch and Hermannsdorfer is still being used, I fancy with less enthusiasm than when it was first introduced. Goldsmith has pointed out that the compound syrup of hypophosphates (British Pharmacopœia) contains the necessary minerals to supplement the diet poor in sodium chloride.[4]

Tuberculosis cutis verrucosa is not rare in Western Australia, but I have seen it only in males. Two patients recently seen were a veterinary surgeon and a labourer at a country sanitary depot. I have been impressed by the early glandular involvement, which was probably aided by pyogenic infection. Is it improbable that a filter-passing virus as a superinfection may explain the tendency to warty growth?

Tuberculosis colliquativa is not uncommon; it responds very well, I find, to treatment with "Elastoplast".

Tuberculosis fungosa cutis is found occasionally on the backs of the hands and forearms of elderly people. It is difficult to distinguish this condition from pyogenic warty conditions following splinter embedding, except through the bacteriological finding, and I believe that a filter-passing virus is common to most of these fungating tumours.

Tuberculosis cutis lichenoides is generally accepted as being tuberculous, and calls for no special discussion.

Tuberculosis papulonecrotica is also typical. Occasionally apparently typical manifestations are found in which there is no evidence of the tubercle bacillus, but the streptococcus is found in the lesions, in the blood and *post mortem*.[5]

Some of the large, indolent, acneiform lesions we see in pale adolescents would, I think, be regarded as "tuberculous", at least in Vienna. Ramel classifies them as gummatous tuberculides with a predilection for the seborrhœic regions.[6]

Erythema induratum, although regarded generally as typically tuberculous, may be mentioned here because it lies at one end of a series of tumours which resemble one another closely.

The sarcoid of Darier-Roussy is regarded as being closely allied to it, and to this condition in turn some chronic types of *erythema nodosum* bear a close resemblance.

The patients with *erythema induratum* seen by me in Australia have impressed me with their otherwise buxom good health and the absence of past tuberculous history. Whitfield[7] describes the type of *erythema induratum* which is not tuberculous, which affects neurotic females about the climacteric who have a rheumatic history; here circulatory disabilities and thin stockings are unhappily combined.

In Class III in Funk's classification are the diseases of uncertain origin; he includes the benign miliary lupoid of Boeck and *lupus pernio*. These manifestations are rare with us and need not be discussed in detail; I shall, however, mention that mucous membranes, lymph glands, eyes, lungs and parenchymatous organs may be all involved with the skin. Bone marrow and bone itself may be attacked. Funk excludes the sarcoids of Darier-Roussy, which presumably are placed in the definitely "tuberculous" class. The general opinion is that a tuberculous ætiology of the sarcoid of Boeck is probable. It is here that one is thrown into the battle of authorities. Some consider these conditions definitely "tuberculous"; others consider that they represent unspecific reactions to several factors; and a third group consider that the condition arises from a specific infection, which is not tuberculous. These conditions occur infrequently with us, I think; but they require some notice, as they represent the *pons asinorum* of cutaneous

allergy. Their importance lies in the fact that in their grouping some are classified with diseases accepted as tuberculous. *Granuloma annulare* is sometimes included with, sometimes excluded from, this group.

Microscopically, sarcoids present a fairly true tubercle formation. The failure to demonstrate the actual bacilli in their structure is in the main recognized; they give in the main no local reaction to tuberculin, and their hosts usually show a reaction to tuberculin at least less than that of controls who are presumably normal.

It would be impossible to quote even a portion of the literature on sarcoids and their allies here.

The question of the "-ids" may now be mentioned. Hallopeau's theory, to which Boeck formerly subscribed, was that tuberculides and the sarcoids *et cetera* were due to the action of toxins derived from a focus of actual infection in some other part of the body. This theory is discredited generally, although a similar one is used to explain the cutaneous action of bromine, iodine and phenolphthalein.

Recently a view sponsored by Dittrich,[8] Gougerot[9] and Memmisheimer[10] has found favour: that a cutaneous tuberculous lesion, by its evolution and treatment, produces some substance which by absorption assists the individual's resistance to pulmonary tuberculosis. It seems hardly logical to accept this view and to deny that of Hallopeau. The theory of a filtrable type of virus of tuberculosis has not had sufficient proof to gain any measure of support.

Darier and Haury advance the theory that dead or attenuated bacilli are responsible. Against this is the occasional production of virulent lesions in the guinea-pig from tuberculide inoculation.

It is generally accepted that every tuberculide is due to the presence of bacilli, (*a*) in small numbers, (*b*) acting against the highly sensitized skin, (*c*) being dispersed by the skin so quickly that their numbers are not great enough to produce microscopic evidence. Jadassohn and Lewandowsky postulate that whenever microorganisms are being repeatedly destroyed by the immunizing processes of the disease (here the skin), tuberculoid and epithelioid processes may result. Kyrle's theory that every sarcoid will contain tubercle bacilli if examined early enough has not, I think, adequate support from his one case, striking though it was.

Recent work by F. R. Sabin[11] indicates that the epithelioid picture is due to lipoid and wax-like substances found in large amounts in all acid-fast organisms, and that the protein proportion of the bacillus is responsible for capillary changes and tissue necrosis. Ray and Shipman[12] state that similar reactions have been obtained by injections of grass derivatives and of colon bacilli. Jordon and Osborne,[13] in a recent paper on Besnier-Boecke's disease, conclude that sarcoid lesions depend on two factors: (i) the presence of insoluble substances, such as lipoids and waxes, present in certain organisms in the living and dead state, (ii) the absence of tissue reaction to other components of the organism. They also state that Besnier-Boecke's disease is not specific, but demonstrably due to two causes, tuberculosis and leprosy.

Sulzberger, in a discussion following this paper, states that some focus of tuberculosis is present in most instances of sarcoid; that the fatty and lipoid reaction does not explain the sarcoid but represents a foreign body reaction.[14] W. Jadassohn has shown that injection of tubercle bacilli into a non-susceptible animal like the rat produces a sarcoid reaction. In the susceptible guinea-pig the reaction is of a different type, and there is no sarcoid unless the guinea-pig has been previously immunized.

Granuloma annulare is considered variously by different authors. Probably the consensus of opinion accepts it as being of tuberculous origin. In most instances in the last two years I have found *granuloma annulare* lesions on the hands or feet. The probability of external inoculation must be considered. Two patients stated that bites by small sea lice precipitated their lesions. An allergic state undoubtedly exists after an attack of tuberculosis; one of my patients previously suffered from pulmonary tuberculosis and another died a year later from the same disease. Again, arguments for and against a tuberculous origin have been produced. The tuberculous reaction has been quoted as "negative" in most cases, and the condition has been described as an anergic state. Personally, I have found a positive reaction in most of my patients.

Against the tuberculous theory is the fact that an epithelioid reaction can be produced by other organisms than the tubercle bacillus, that animal inoculation proves nothing, while the administration of arsenic has a beneficial effect on the lesions.

The same may be said of the action of X rays and of supersoft X rays.

I have a theory, which I submit with due humility: it is that in the struggle of the individual against tuberculous invasion an excitation of the reticulo-endothelial system occurs and may last for years. This can be manifested as a tendency to local cellular proliferation in response to other irritants as well as the tubercle bacillus. If this theory is acceptable it will bridge over the divergent opinions as to the specific nature of the granulomata mentioned.

Many of you will remember the controversy some years ago as to the origin of *erythema nodosum*, and the stout championship by the late Dr. A. A. Lendon of "nodal fever" as a disease *sui generis*. Scandinavia accepts *erythema nodosum* as of tuberculous origin, although the German idea is generally opposed to this. Ramel, of Lausanne, states that in *erythema multiforme* as well, tubercle bacilli can be demonstrated.[6] Barber, in his Lettsomian Lecture in 1929,[15] considered *erythema nodosum* not as a disease, but as a skin reaction to certain types of stimuli in sensitized individuals. J. Odery Symes[16] in 1932 gave figures to show the frequency

with which pulmonary tuberculosis occurred as a sequel to *erythema nodosum* (in about 10% of cases). This has been countered by a suggestion that *erythema nodosum* acts by "stepping up" in latent tuberculous infection, as occurs sometimes after measles. Embery claims that it is an autogenous tuberculous reaction; others regard it as a transient state in which the tuberculous reaction is changing from negative to positive.

It may be generally concluded: (i) that some atypical, long-drawn-out forms closely approximate tuberculides; (ii) that the reaction may be produced by organisms other than the tubercle bacillus; (iii) that if one allows for varying degrees of immunity and resistance in the same individual, it is easy to accept *erythema nodosum* as an occasional manifestation of systemic tuberculosis; (iv) that in the majority of cases *erythema nodosum* causes no tuberculous manifestations previously or subsequently.

Lupus erythematosus concerns us more closely than most of the previously mentioned diseases. It is generally acknowledged that its frequency is on the increase whilst its cause still remains obscure. There has been a tendency of late years to consider the acute generalized form as a disease distinct from the fixed discoid type. This, I think, is not justified, as undoubted instances occur in which there is an alternation of types. In France, Austria and Switzerland a tuberculous ætiology is accepted; in Germany the condition is regarded as having a multiple ætiology; and in England streptococcal infection is regarded as the usual cause.

Keil,[17] from a study of 125 cases from literature, found that only 20% of patients with *lupus erythematosus* had active or possibly active tuberculosis, and that three-fourths of these 20% were of Continental origin. In 20% of cases inactive tuberculosis was found, and in 50% there was no evidence of tuberculosis at all. He deduced from this that tuberculosis was coincident and unrelated.

Positive results from guinea-pig inoculation have been reported by Gugerot in 1908, by Ehman and Remes (1908) and by Bloch and Fuchs (1913).

Darier states, without giving the authority, that the large glands found in connexion with *lupus erythematosus* are bacilliferous and inoculable.

In America multiple ætiological factors are considered, and the frequent associations with papulonecrotic tuberculosis, sarcoid *erythema induratum et cetera* make its origin suspect. That previous or coexisting tuberculosis increases the skin allergy must, I think, be generally accepted.

Acute *lupus erythematosus* has been reviewed by Mook, Weiss and Bromberg,[18] and by F. Engman, junior,[19] The latter concludes that early *lupus erythematosus* is a constitutional disease, in which the erythema is entirely secondary and perhaps not present, at least in the early stages.

I had some years ago the opportunity of observing in a young married woman, first, an active generalized erythema, which on subsiding left patches of discoid *lupus erythematosus*. Some months later, while under treatment with "Solganol", she contracted a generalized rash, and less than a year after that, and three days before the birth of her first child a third eruption occurred. I was unable clinically to distinguish any differences between these rashes.

Stokes[20] regards acute *lupus erythematosus* as entirely an allergic condition, and he suggests that the condition should be treated with rest and autohæmotherapy. In severe cases he recommends blood injections from a donor previously given injections of gold. Stokes regards influenza as "stepping up" the process.

Sulzberger suggests that some cases are due to the use of drugs taken in various prepared foods or medicinally.

Keil[21] regards systemic *lupus erythematosus* as connected with a form of thrombocytopenic purpura. He states that its incidence falls on the smaller vessels, particularly on the capillaries, and that hyaline thrombi are characteristic findings. This suggests some infection common to both conditions.

O'Leary states that Welsh, at the Mayo Clinic, has isolated a streptococcus in *lupus erythematosus*. Arndt claims to have found tubercle bacilli in fixed patches of *lupus erythematosus*; Kren and Löwenstein claim to have found them in blood culture in 100% of cases. Bloch and Fuhs (quoted by Goldsmith) have, by injection of filtered extracts of *lupus erythematosus* skin, produced a tuberculin-like reaction in tuberculous patients.[22] Gray regards *lupus erythematosus* as due to a single cause, and that is not tuberculous.

The streptococcal theory has by no means been definitely proved.

The tuberculous connexion with *lupus erythematosus* is thus generally in dispute. It is in my own feeling that a previous tuberculous infection has "stepped up" the allergic reaction in these patients; it is difficult to prove, but if one regards the chronically enlarged glands in the neck and axillæ as probably tuberculous, I find them much more in evidence in *lupus erythematosus* than in apparently normal people. Now that tuberculous glands of the neck are being treated more conservatively, there is an interesting study for the future as to what proportion of patients with *lupus erythematosus* will give a history of having had such glands.

In conclusion, may I draw attention to the terrain in *lupus erythematosus*. It is known that the chief incidence is in the smaller vessels, particularly the capillaries. It is precisely those regions where capillary reactions have been predominant that *lupus erythematosus* selects. The cheeks and ears throughout childhood, adolescence, maturity and senility react constantly to physical, mental and probably hormonic influences. Incidentally one notes the predilection of *acne rosacea* for the same area. You are all familiar with the rough patches on the faces of young children, which Sabouraud still maintains are due to the streptococcus; hence *impetigo sicca*. Years ago, Whitfield, in disagreeing with Sabouraud, attributed the condition to

dribbling and pillow-wetting by children with naso-pharyngeal disabilities. The use of certain soaps, of course, we all know to be a frequent factor.

However, there is much more to it than this. The condition persists long after the dribbling age, and it is definite that (*a*) these rough patches are in constant association with nasal pharyngeal infection and catarrh, and (*b*) that a catarrhal attack is invariably followed by a lighting up of the areas. This, in my opinion, is an allergic reaction.

It is interesting to speculate whether these children, allergic in youth, represent candidates for the allergic capillary reactions of *lupus erythematosus* in later life. Light, of course, we all recognize as being an important factor in this disease; that it is not the only factor is shown by the cases in which the disease commences and is confined entirely to the hairy scalp.

There is one more suggestion: that either an allergic reaction or a deficiency caused by a previous disease may give rise to a circulating toxin with light sensitivity, a mechanism we believe to be at work in pellagra. Scholtz,[23] in a discussion on Jordon and Osborne's paper, suggests a more dynamic view of cutaneous tuberculosis, and less hair-splitting in diagnosis. He suggests that one form of cutaneous tuberculosis may change into another according to time and the resistance of the individual.

We are all, I think, inclined to be too static in our concept of diseases; a microscopic slide may suggest a beautiful picture of still life, but life is never still.

References.

(1) B. Dragisić: *"Granulosis rubra Nasi im Kindesalter als Teilsymptom der infantilen Akrodynie"*, *Dermatologische Wochenschrift*, Volume CIV, April 10, 1937, page 461.
(2) Darier: *"Précis de Dermatologie"*.
(3) H. Fischer: *"Die Behandlung der Lupus Vulgaris mit verdauenden Mitteln"*, *Dermatologische Wochenschrift*, Volume CIV, March 13, 1937, page 337.
(4) W. N. Goldsmith: "Recent Advances in Dermatology", 1936.
(5) W. W. Duemling: "Dermatitis Nodularis Necrotica: Report of a Case and Review of Literature", *Archives of Dermatology and Syphilology*, Volume XXI, February, 1930, page 229.
(6) E. Ramel: "Some Observations on Causation of Erythema Exudativum Multiforme (Hebra)", *British Journal of Dermatology*, Volume XLII, January, 1930, page 1.
(7) Whitfield: "Skin Diseases and their Treatment".
(8) O. Dittrich: *"Lichen Zoniformis Syphiliticus"*, *Dermatologische Zeitschrift*, Volume LXXII, January, 1936, page 305.
(9) H. Gougerot: Deliberations of the International Congress of Dermatology, 1935.
(10) A. Memmesheimer: *"Untersuchungen über den Einfluss der Hauttuberkulose auf die Lungentuberkulose"*, *Archiv für Dermatologie und Syphilis*, Volume CLXXIV, 1936, page 511.
(11) F. R. Sabin: "Cellular Studies in Tuberculosis", *American Review of Tuberculosis*, Volume XXV, February, 1932, page 153.
(12) L. W. Ray and J. S. Shipman: "Tissue Changes Produced by the Action of the Lipoids of Tubercle, Grass and Colon Bacilli and of Liver", *American Review of Tuberculosis*, Volume VII, 1923-1924, page 88.
(13) J. W. Jordon and E. D. Osborne: "Besnier-Boeck's Disease: Report of Two Cases of Extensive Involvement", *Archives of Dermatology and Syphilology*, Volume XXXV, April, 1937, page 663.
(14) Sulzberger: *Archives of Dermatology and Syphilology*, Volume XXXV, April, 1937.
(15) H. W. Barber: "Lettsomian Lectures on the Relationship of Dermatology to other Branches of Medicine", *The Lancet*, Volume II, 1929, August 24, page 363; September 7, page 483; September 21, page 591.
(16) J. O. Symes: "Significance of Erythema Nodosum", *British Journal of Dermatology*, Volume XLIV, April, 1932, page 181.
(17) H. Keil: "Relationship between Lupus Erythematosus and Tuberculosis: Critical Review based on Observations at Necropsy", *Archives of Dermatology and Syphilology*, Volume XXVIII, December, 1933, page 765.
(18) W. H. Mook, R. S. Weiss and L. K. Bromberg: "Lupus Erythematosus Disseminatus", *Archives of Dermatology and Syphilology*, Volume XXIV, November, 1931, page 786.
(19) M. F. Engman, junior: "Early Acute Lupus Erythematosus", *Archives of Dermatology and Syphilology*, Volume XXXV, April, 1937, page 685.
(20) Stokes: *Archives of Dermatology and Syphilology*, Volume XXXV, April, 1937.
(21) H. Keil: "Relation between 'Systemic' Lupus Erythematosus and a Peculiar Form of Thrombocytopenic Purpura", *The British Journal of Dermatology*, Volume XLIX, May, 1937, page 221.
(22) W. N. Goldsmith: "Recent Advances in Dermatology", 1936.
(23) M. Scholtz: "Dynamics of Cutaneous Morphology: An Analytic Study", *Archives of Dermatology and Syphilology*, Volume XXXIII, April, 1936, page 605.

SOME EXPERIENCES OF PROSTATIC SURGERY IN A PROVINCIAL CITY: A REPORT OF ONE HUNDRED AND NINE CASES.[1]

By G. R. DAVIDSON, M.B., F.R.C.S. (Edinburgh), F.R.A.C.S.,
Honorary Urologist, Ballarat Hospital, Victoria.

THIS paper is based on the results of the investigations and treatment of a first series of 109 cases of non-malignant prostatic obstruction, carried out mainly during the past seven years. I propose, before discussing in some detail the methods of treatment that are carried out at the present time in the Ballarat Hospital, to mention very briefly a few points regarding the anatomy and pathology of the normal and obstructing prostate gland.

Anatomy.

The prostate gland, which is described as having a base, apex, anterior, posterior and two lateral surfaces, lies with its base beneath the floor of the bladder and its apex at the triangular ligament. It is pierced by the prostatic urethra, which is not placed centrally, but which lies anterior to the central axis of the gland; and it is also pierced by the two ejaculatory ducts surrounded by a sheath of non-striped muscle. These structures pass upwards and backwards from the verumontanum through the sheet of gland tissue which unites the two lateral lobes behind the urethra, appearing in a vertical median section to isolate a wedge of gland, thus forming the so-called median lobe.

For descriptive purposes the prostate may be regarded as being formed by the fusion of five lobes: a small anterior lobe in front of the urethra, two lateral lobes, a middle lobe lying between the urethra and the ejaculatory ducts, and a posterior lobe lying between the ejaculatory ducts and the rectum. It should be remembered that the prostatic lobes are fused and the boundaries are obliterated in the adult. The pathological middle lobe should be differentiated from the embryological middle lobe. According to Lowsley,[1] and without entering into detail, the prostate develops in intrauterine life by the outgrowth of five buds of tubules in the posterior part of the urethra. In the anterior lobe the glandular elements are few, and begin to shrink early in development. Before adult life is reached,

[1] Read at a meeting of the Victorian Branch of the British Medical Association on February 2, 1938.

this lobe in the majority of persons has atrophied. Very occasionally a few tubules persist and can on rare occasions cause an enlargement.

During development the middle and lateral lobes tend to fuse, but the posterior lobe is separated from them and from the ejaculatory ducts by fibrous interlobar partitions. It forms the posterior surface of the gland and is, of course, the lobe mainly felt on rectal examination. It is separated from the rectum by the recto-vesical fascia, or fascia of Denonvilliers, which is formed during developmental life by an obliteration of the recto-vesical pouch of peritoneum. This fascia, which is an important surgical landmark in perineal prostatectomy, tends to limit the spread of carcinoma from prostate to rectum and *vice versa*. It forms the posterior surface of the so-called prostatic sheath. Occasionally, as a developmental error, the recto-vesical pouch may fail to become obliterated satisfactorily, and extending nearly to the anus may contain loops of bowel. It is obvious that disastrous results would follow in such a case from the misdirected and over-enthusiastic use of a prostatic resectoscope.

Occupying a very strategic position from the point of view of obstructive possibilities are the subcervical and subtrigonal glands, which lie between the mucous membrane of the trigone and posterior part of the urethra. These glands are of great surgical importance, because they, like the prostatic elements, are prone to undergo degenerative changes associated with enlargement in middle and old age. It is therefore obvious that the symptoms of so-called prostatic obstruction may arise from an enlargement of either the prostate or the submucosal glands, and according to some authors the relative incidence is approximately equal. Krogius[2] has drawn attention to the histological similarity between the glands of the prostate and these accessory glands.

The greatest diameter of the posterior part of the urethra is one centimetre and its length from the internal urethral orifice to the superior layer of the urogenital diaphragm is three centimetres. It is quite obvious that even minor degrees of encroachment in this particular area will be followed by a considerable disturbance in function, and that similarly the removal of even a trifling amount of tissue will produce a disproportionate degree of relief.

It was dissatisfaction with this small amount of tissue removed at open operation which caused surgeons to look for simpler and safer methods of dealing with this type of obstruction. Young's punch was the first of the more modern instruments to be designed for this purpose, and that was followed in more recent years by the various types of prostatic resectoscopes which are now in use.

The gland tissue of the prostate itself is enclosed in a capsule which consists of layers of fibromuscular tissue continuous with and forming part of the stroma of the organ. When a prostate becomes the site of a benign enlargement (whether that enlargement starts in the submucosal glands or in the prostatic glands proper) it almost inevitably starts next to the urethra, causing a lateral and posterior compression of the prostatic tissue, producing that false capsule out of which the so-called adenomatous masses usually peel so easily. Any attempt to enucleate a normal prostate or a fibrous prostate will end in trouble and will lead mainly to an outpouring of the operator's sweat and the patient's blood.

The extrinsic prostatic sheath is derived from visceral pelvic fascia. Veins lie in the loose areolar tissue between the sheath and the capsule. Between the *symphysis pubis* and the anterior aspect of the prostate is a plexus of veins formed by the union of the dorsal vein of the penis and some of the prostatic veins. This prostatic plexus of Santorini eventually empties into the hypogastric veins.

The arterial supply of the prostate is derived from branches of the middle hæmorrhoidal, inferior vesical and internal pudendal arteries. The most important is the inferior vesical artery, which arises from the anterior division of the hypogastric artery. It terminates in the prostatic artery, the main trunk of which gives off two sets of branches, ascending and descending. The ascending branches enter the base of the prostate and bladder. During the enucleation of the prostate these branches are severed, and this may result in serious hæmorrhage. The postero-lateral hæmostatic sutures in the Harris operation are designed to control hæmorrhage from these arteries.

The Pathology of Bladder Neck Obstruction.

Even at the present day I think we must confess that we are uncertain of the pathology of prostatic enlargement, and even more indefinite as to its ætiology. Any abnormality of the prostate is capable of causing obstruction, but generally speaking there are three main causes.

1. Hyperplasia of the prostate, adenomatous prostate, prostatic hypertrophy are names commonly given to the simple enlarged prostate.

2. The formation of a median bar, contracture of the vesical neck, fibrous prostate, prostatism *sans* prostate are names given to denote that condition in which obstructive symptoms develop without an obvious enlargement of the gland.

3. Carcinoma of the prostate is the third.

Benign Prostatic Enlargement.

The microscopic appearance of an enlarged prostate is one of glandular hyperplasia, associated with fibrous and muscle overgrowth. Very rarely a leiomyoma or pure muscle cell tumour occurs in the prostate. The nodules that arise in the prostate are commonly referred to as adenomata, but the consensus of opinion is that they are rather formed by a hyperplasia of preexisting tissue than of new glandular tissue.

As previously mentioned, the ætiology of the enlarged prostate is obscure and undecided. It has been frequently suggested that it bears some rela-

tionship to gonorrhœa, but as Boyd[3] says, the evidence is altogether too inconclusive to tarnish the character of respectable old gentlemen.

It would be wearisome in the extreme if I attempted to discuss in any detail the various theories regarding the ætiology of prostatic enlargement. Possibly the most modern is that summarized by Hugh Lett[4] in a recent article in *The British Journal of Surgery*. I shall quote his words:

> It seems probable that the male hormone proper is secreted by the interstitial cells of the testis, and that this hormone is the main factor in the development and control of the prostate. Some consider that benign enlargement is due entirely to a diminution in the secretion of this hormone as age advances. Others postulate the presence of a second hormone, arising from the seminiferous tubules, which they call inhibin. They believe that it controls the gonadotropic activity of the pituitary gland, and that as in the age of involution the tubular cells of the testis become diminished in number less inhibin is formed; the pituitary then becomes hyperactive, and as a result there is excessive stimulation of the interstitial cells of the testis, which in their turn produce the prostatic enlargement.

This may be summarized briefly as follows:

Absence of inhibin
|
leads to
|
Pituitary hyperactivity
|
which
|
Stimulates androtin
|
and produces
|
Prostatic enlargement.
(Hinman.[20])

The purified lipoidal extract of a bull's fresh testis has been called "androtin", which is the homologue of "theelin" of the ovary.

This theory apparently breaks down when one seeks to explain by it the question of racial selectivity that undoubtedly exists so far as prostatic enlargement is concerned. Kenneth Walker[6] points out that amongst Mongolians prostatic enlargement is an extremely rare condition. He mentions that reports from the Genito-Urinary Hospital at Kyoto show that there is only an average of four cases of this enlargement *per annum* among many thousand of cases that pass through the department. Surely it is reasonable to suppose that Japanese testicles, like other Japanese organs or articles of manufacture, are affected by the passage of time and the stresses and strains of life. I feel that it is highly probable that someone delivering an address on this subject in this hall in twenty years' time will say: "Gentlemen, the ætiology of benign prostatic enlargement is still rather obscure."

Median Bar.

At the fifth International Congress of Urology, held in London in 1933, urologists from all parts of the world discussed, among other subjects, the surgery of the neck of the bladder. According to *The British Journal of Urology*,[7] twenty urologists spoke, beginning with the famous Professor Marion, of Paris, and ending with the equally famous Hugh Young, of Baltimore. On reading through a summary of the uterrances of the principal speakers and those who discussed the question, one was struck by the complete lack of unanimity, as far as nomenclature was concerned, and also by a lack of detailed agreement as far as pathology was concerned.

Alexander Randall,[8] of Philadelphia, has done an immense amount of work on the various contractures of the bladder neck, grouped under the term "median bar", and it is his classification that I shall briefly bring before you.

He describes: (*a*) the acute "prefibrotic" bar, (*b*) the muscular bar, (*c*) the true median bar, which he subdivides into fibrous and glandular types.

The acute prefibrotic bar is a contracture of the bladder neck, arising from inflammatory changes associated with posterior urethritis and prostatitis. It is probably the initial stage of a chronic fibrous bar. This condition is unsuitable for resection, as it responds as a rule to treatment of the underlying urethritis and prostatitis.

The muscular bar is the term used to describe a congenital hypertrophy of the bladder neck. Symptoms may occur at almost any stage in life, but essentially there has been some difficulty of micturition ever since infancy.

The true median bar may be either fibrous or glandular. The fibrous type, which is the end result of infection, may be either intraurethral or intravesical, depending on whether it shortens or lengthens the posterior part of the urethra.

The glandular type of median bar is really an early stage of prostatic hyperplasia, occurring in glands of the posterior commissure of the prostate, under the muscle of the internal sphincter and within the prostatic capsule. When this enlargement occurs, it produces a thick round-edged bar. Although differing in position, it is similar in nature to that enlargement of the subcervical and subtrigonal glands previously mentioned.

The Author's Series.

This paper, as mentioned previously, is primarily based on the results of operations in 109 cases of benign prostatic enlargement or bladder neck obstruction. Seventy-seven patients have been treated by open suprapubic operation with a mortality of four. The criterion of recovery from operation has been that the patient should be well enough to leave hospital, micturating normally, and that his wound should be healed.

I have not included as deaths due to operation three cases that I shall mention.

> The first patient, after being up and about for nearly two weeks, became feverish the day he was due to leave hospital, over a month after operation. He contracted a respiratory infection and later pneumonia, from which he died.

The second died during his convalescence from an intestinal obstruction secondary to an unsuspected malignant growth in the bowel. He was up and micturating normally before this happened.

The third patient, who had gross pyelonephritis, developed a leak from his wound some months after operation. This was repaired without difficulty, but he gradually became weaker and died of uræmia.

The majority of the suprapubic operations have been performed for the removal of so-called prostatic adenomata, and whenever possible this has been done in one stage, which is obviously an advantage. No patients are catheterized in the casualty department of the Ballarat Hospital; they are admitted directly to the wards for treatment, and the following routine is carried out.

After the patient has had a bath, the anterior part of the urethra is washed out with a weak antiseptic solution, such as oxycyanide of mercury, 1 in 5,000. A rubber catheter, with a Coudé tip, is passed in the usual manner. A small piece of gauze soaked in flavine, 1 in 1,000, is wrapped around the catheter where it emerges from the urethra, and the catheter is held in position with strips of adhesive plaster. If the bladder is over-distended, gradual decompression is carried out. A simple and effective method is to plug the end of the catheter with a spigot or with a sterile cork, and to push a small bore hypodermic needle through the rubber wall of the catheter, allowing it to drip into a sterile dish placed between the patient's thighs. Later the catheter is connected by sterile tubing to a reservoir tied to the bed, which contains an antiseptic, such as "Cyllin", and the end of the tubing is always kept in this antiseptic solution. Daily bladder irrigations are given, their number depending on the degree of infection present. In accordance with the teaching of Harris,[9] the bladder, when infected, is irrigated with potassium permanganate solution, 1 in 8,000, then with plain sterile water, and finally four ounces of a 1 in 3,000 solution of silver nitrate are instilled and held in the bladder for thirty minutes. Sometimes it is necessary to increase the strength of the silver nitrate solution up to 1 in 1,500 in the more grossly infected bladders. During this period, urinary acidifiers, such as ammonium chloride, sodium acid phosphate, or nitrohydrochloric acid, are prescribed, and hexamine, in the absence of hæmaturia, is given with any of these. On this treatment the majority of infected bladders tend to clear up very well. If the infection proves unduly stubborn, there is, I think, usually some other factor or factors present, such as infection higher in the urinary tract, for example pyelonephritis, or some condition in the bladder, such as a stone or a diverticulum. While this period of bladder drainage is being carried out, the patient is encouraged, whenever possible, to get out of bed and to move about with the catheter *in situ*. In contradistinction to the sentiments expressed in the popular song about the gentleman who was "dead but wouldn't lie down", prostatic patients very often tend to be dead if they will not get up. I regard it as important to keep these old men moving. The catheter is changed every third day, being reinserted after the patient has had a bath and after the urethra has been irrigated.

During this stage of preparation, such conditions as chronic bronchitis, dental sepsis, constipation *et cetera* are treated, and also notice is taken of the patient's mental outlook, which is important, because impairment of mental function is not uncommon and quite frequently is an indication of latent uræmia. Patients who have been operated on and who are expected to have an uneventful convalescence are placed, whenever possible, on their return from the theatre next to or near a patient who may be awaiting operation. If the patient is not expected to do well he is returned into a smaller and quieter ward.

Patients are encouraged to drink as much water as possible, and will often drink from five to ten pints a day. An excessive water consumption sometimes seems to produce nausea and anorexia, which is only temporary, disappearing when the excess of fluid is cut down. I think that internal lavage of the urinary tract is of great importance.

When a catheter is introduced into a urethra, no matter what precautions are taken, a certain degree of urethritis will result, which usually manifests itself on the second or third day. Sterile residual urine almost always becomes infected to some degree from catheter drainage, and the patient develops, I find, quite frequently an acute urinary infection. For that reason one should, if it is at all possible, avoid catheter drainage in patients with sterile urine; and when it is necessary, as it nearly always is, no trauma, whether in the nature of an enucleation or a resection, should be carried out until at least a week or ten days have elapsed.

I feel happier about operating on a patient who has had some degree of bladder sepsis which has responded to treatment, than on one who has come into hospital with probably sterile urine and whose bladder has been drained by an indwelling catheter for a week or so. In the former case the infection has probably been present for months and the patient has had time to build up a resistance against his own organisms. Infection, however, is all a matter of degree, and patients with residual urine already infected are highly intolerant of any superadded sepsis. J. Gray,[10] writing in *The British Journal of Urology* in 1932, stated that he picked out 22 cases at random from the wards of Saint Peter's Hospital during a period of twelve months. The only factor influencing selection was that every patient had sterile urine prior to examination and treatment. He stated that out of the 22 patients investigated none escaped infection, which occurred after either instrumentation or operation. There were five deaths in the series, a mortality of 23%. He compares those figures with the general mortality rate of all patients with enlarged prostate who had a chronic infection of the urine prior to operation, which was during that year 10%. He suggests that operation in these patients, who are

really acutely infected, carries the heavier mortality and higher incidence of complications.

Renal Function Tests.—When the patient's appearance indicates his general improvement, renal function tests are carried out. MacLean's urea concentration test has been used until recently and has proved a simple but only a fairly reliable test.

Lately a modification of the phenolsulphonepthalein test, described by R. J. Silverton,[11] of Sydney, has been used. In this test, one cubic centimetre of the dye is injected intramuscularly. The urine is collected for seventy minutes into a sterile bottle, and this is marked (1); it is then collected for sixty minutes, and this is marked (2). The reason why seventy minutes' drainage is allowed for specimen (1), as against sixty minutes for specimen (2), is that it takes approximately ten minutes for the dye, when injected intramuscularly into a normal person, to commence being excreted by the kidneys. Each specimen is then diluted with water up to one pint and is made alkaline with *liquor potassæ*. Samples are taken in specimen glasses of equal size and held up to the light and compared. A normal kidney should excrete much more strongly in the first hour than in the second. Thus the first glass will present at least a medium magenta colour, while the second should be a lighter pink. When renal efficiency is impaired there may be no colour in the first glass and only a trace of pink in the second. Intermediate degrees of lowered renal function will show corresponding variations in the colour of the two glasses. An estimation of the blood urea is not necessary when the result of the phenolsulphonepthalein test is within normal limits. When the latter is low, however, it is of some value, because a low figure for the phenolsulphonepthalein test without retention of urea in the blood indicates a lesser degree of insufficiency than such a result to the test with retention of urea; and the amount of increase in the blood urea is a further guide to the degree of renal failure.

The indigo-carmine test has been recommended by Harris,[12] and obviously in his hands must have been satisfactory. He was satisfied if an excretion of the dye occurred under seven minutes. It has seemed to me that when there is a big post-prostatic pouch the first traces of the dye will not drain out at once, and if water is run in to wash this out the dye may be so diluted as to be not very obvious. According to Hinman,[13] the dye may be excreted decolorized. He considers that phenolsulphonepthalein is superior to indigo-carmine in every way.

Tests evolved for the estimation of renal function have been many and varied, and the sensible application of one or more tests, preferably simple, is of course essential. However, clinical appreciation of the patient's general condition is of the utmost importance. Such factors as what he was able to do before his admission to hospital, the condition of his tongue, his appetite, his ability to sleep, his zest for life, must all be taken into consideration.

Anæsthesia.—About 70% of the present series of operations have been carried out under some form of local anæsthesia. For the last two or three years practically nothing but spinal anæsthesia has been given, a heavy solution of "Percaine" being used. Before that, anæsthesia was induced by caudal and transsacral injection in association with a subumbilical field block, "Novocain" being used. The anæsthesia thus obtained was satisfactory, but the procedure was time-consuming and rather difficult. The use of the heavy solution of "Percaine" has given good results, and up to the present I see no indication to change to any other form of anæsthesia. R. J. Silverton, of Sydney, uses this solution extensively in his work, and I am greatly indebted to him for demonstrations of his technique.

I would summarize the advantages of spinal anæsthesia as follows:

1. In hospital practice the majority of anæsthetics are given by the resident medical staff, who are frequently changing, with consequent changes in the quality of the anæsthetic given. Spinal anæsthesia is constant.

2. Despite claims to the contrary, I think that pulmonary complications are probably less after spinal anæsthesia. Unless the patient suffers unduly from shock, he is encouraged to take deep breaths within an hour or so of the operation. Chevassu,[14] in speaking of anæsthesia in urology, condemns inhalation anæsthesia, and he considers that many post-operative sudden deaths which are attributed to emboli are in fact instances of latent uræmia to which the general anæsthetic has been a contributory factor.

3. Muscular relaxation is excellent.

4. "Percaine" appears to produce in the majority of cases a prolonged period of analgesia, which makes the previously mentioned efforts at chest expansion easier and lessens the amount of post-operative sedation necessary.

5. Operations performed under spinal anæsthesia are associated with less bleeding than those carried out under inhalation anæsthesia.

Malcolm Baillie,[15] working at King's College Hospital, carried out an investigation on the amount of blood lost during the operation of prostatic electroresection in 50 consecutive cases.

He showed that the average blood loss under inhalation anæsthesia was 3·25 ounces, while under spinal anæsthesia it was only 1·48. Incidentally it might be mentioned here that the average blood loss occurring in suprapubic prostatectomy is about half a pint.

The chief danger and drawback to spinal anæsthesia is shock, which is mainly due to lowered blood pressure, but which may also be contributed to by the nervous susceptibility of the patient. To a certain extent the latter may be controlled by pre-operative sedation. Injections of morphine, one-sixth to one-quarter of a grain, and hyoscine, one two-hundredth to one one-hundredth of a grain, are

given two hours and one hour before operation. The fall in blood pressure may be controlled to a certain extent by the administration of pressor substances, such as ephedrine, before and during operation, and adrenaline during operation, and also by the administration of the least possible amount of "Percaine" sufficient to cause satisfactory anæsthesia.

Blood pressure readings are taken throughout the operation and charts are kept. It has been noticed that there is almost inevitably a fall of blood pressure simultaneous with the enucleation of the prostate, although the anæsthesia is perfect. This appears to be quite apart from the normal fall in blood pressure associated with the anæsthetic. I am at a loss to explain why this occurs. Possibly some depressor substance may be squeezed into the circulation while the gland is only partially enucleated. Spinal anæsthesia should be avoided in men with systolic blood pressures below 110 millimetres of mercury, and also in those who show signs of marked vascular or organic nervous disease.

Operative Technique.—For the last three years or so I have been endeavouring to carry out the Harris technique with certain slight modifications, the essential modification being that the bladder is not completely closed, a small self-retaining catheter being left in the suprapubic wound for from four to five days. I do not feel that this is a disadvantage. In a large proportion of cases the tissues fall together after the removal of the suprapubic tube, and the urethral catheter is then sufficient for drainage. Generally speaking the wound has practically healed in a fortnight and natural micturition is taking place. The essential features of the Harris operation in my opinion are the positive control of hæmorrhage, the obliteration of dead spaces and the retrigonization of the prostatic cavity. Complete closure is by comparison a secondary consideration.

Although I use the small transverse skin incision recommended by Harris, I find that in the majority of cases I am able to enucleate the prostate without the aid of a finger in the rectum. I think the added muscular relaxation of the spinal anæsthetic helps considerably in this. It is certainly advantageous to be able to use the forefinger of the left hand in some enucleations, and I feel that no useful purpose will be served by having it imprisoned in a septic cavity like the rectum unless there is absolute necessity for it.

The late Sir John Thompson-Walker told me that in his opinion there was more likelihood of embolism, owing to damage of the veins lying between the capsule and sheath, in operations in which a finger in the rectum was used. I mention this because I have not seen it written anywhere.

The actual details of the operation have been described so frequently and so fully that it is unnecessary for me to do more than mention a few of the more essential features. To my mind these are:

1. The exposure and incision of the empty bladder. The fact that the bladder is irrigated and emptied before operation does not add materially to the difficulties and it is certainly an important factor in obtaining a clean wound.
2. The intraurethral enucleation of the prostate, as opposed to an enucleation commencing at the periphery of the gland. The latter method removes far more of the mucous membrane of the posterior portion of the urethra than the former does.
3. The visualization of the ureteric orifice before the insertion of the postero-lateral hæmostatic sutures.
4. The passage of the retrigonization suture. This is the most important point in the operation. Again when the ureteric orifices are visualized, the needle point should be entered well behind the ureteric bar. A mistake I made for a long time was to tend to depress the handle of the needle holder too soon. Keeping the point of the needle parallel to the mucous membrane of the bladder, I now endeavour to push it down as far as possible under the prostatic cavity before depressing the handle.
5. The pushing of the knot of the retrigonization suture down as far as possible with the tip of the forefinger of the left hand. (Provided the operator is standing on the left side of the patient, which is recommended.)
6. The embracing of a wide amount of tissue by the anterior transverse sutures. They should be tied before the catheter is passed, and not tied too tightly.
7. The retention of the catheter in position by a suprapubic stitch.

On occasions cases are encountered in which it is inadvisable to carry out the full procedure outlined above. I refer to the borderline patient, the "poor risk", with a prostate that is difficult to remove, perhaps with calculi in the bladder, almost certainly with chronic pyelonpehritis and myocarditis. In operating on these men time is the essence of the contract. I feel that unless one is a very skilled and rapid operator, the less one attempts, the better. Realizing the truth of the ancient adage about the undoubted superiority of a live mouse over a dead lion, I content myself with a rapid inspection of the prostatic cavity, which, if it is free from tags of mucous membrane *et cetera*, is packed, if the degree of hæmorrhage necessitates it, with gauze. This gauze, which is removed on the fourth day, is brought out beside the suprapubic tube, not through it. I should like to say a word here in favour of suction drainage, which I employ in cases in which a suprapubic drainage tube is used. This suction drainage is laid on to the wards, and it is a simple procedure to connect it, via a drainage bottle, to a catheter which is inserted into the suprapubic tube. The wound is kept very dry, the patient is comfortable, and, provided the method is used intelligently, I fail to see any drawback to it.

A bilateral vas section is performed at the time of operation and has so far successfully prevented epididymitis.

Transurethral Resection.—Regarding transurethral resection, I have been practising this method of treatment for the past three years. The instrument used has been a Canny Ryall's modification of McCarthy's urethrotome, which has been activated by a spark gap type of machine supplied by Stanford. I imagined at first that prostatectomy could be replaced by a simple and less drastic procedure, one that was associated with a minor degree of risk and a short convalescence. I soon realized that I was being unduly optimistic and that the method was one associated with special difficulties and risks. Patience, judgement and a highly developed technique are necessary for success, and I feel certain that it is only by performing this operation very many times that one can ever hope to become a resectionist of the first order.

At present I regard transurethral resection as the operation of choice in cases of obstruction by median bar, fibrous prostate and sclerosed bladder neck, and in some cases of carcinoma of the prostate. Small middle and lateral lobe enlargements respond well, and quite frequently one feels that it is the procedure of choice when the enlargements are more pronounced. I refer to the elderly or enfeebled man, perhaps with chronic bronchitis or a failing heart. I feel, if one is prepared to exercise due care and patience and is also prepared to repeat the process on more than one occasion, that resection holds out better hopes than a suprapubic removal.

The following case is one in which it was obviously impossible to carry out any surgery by the suprapubic route.

The old man suffered from complete retention of urine due to a combined lateral and middle lobe enlargement. As well as a large incisional hernia, he had myocarditis associated with chronic bronchitis and also a partially ankylosed left hip. Using low spinal anæsthesia I was able to resect between thirty and forty strips, averaging about an inch in length, from the middle and lateral lobes of his prostate. He was able to micturate, according to himself, normally six days after operation.

Probably insufficient has been cut away, and I have advised him to report for reexamination and possibly further resection.

During the actual operative procedure it is obviously of the greatest importance that the cutting loop should be localized at all times. It is quite obvious that any loss of control of the activated loop may lead to damage of such structures as the bladder wall, rectum or external urinary sphincter. The anterior portion of the bladder neck is seldom if ever distorted; therefore, if the operator is uncertain of his position, that area is a safe landmark. The distal landmark is the verumontanum; and if one does not cut below that, no damage to the external sphincter should result.

Hæmorrhage and sepsis are the two main complications associated with this procedure. Regarding the control of hæmorrhage during operation, it is probably wisest to coagulate each individual bleeder as it occurs, at the same time remembering that excessive coagulation around the bladder neck will later lead to the formation of slough and possible secondary hæmorrhage.

Bleeding that is more difficult to deal with may be controlled by the use of a Foley's bag. This small soft bag is built around a catheter which is passed in the usual way. The bag is then inflated with water and traction is exerted, the soft bulbous swelling being pulled against the hæmorrhaging area.

Infection is best dealt with by adequate preoperative treatment.

N. G. Alcock[16] states that in his opinion a surgeon does not become a resectionist until he has operated on at least 100 patients. His own mortality rate was 20% in his first 75 cases, and a study of other series shows that the mortality figures for the first 50 to 100 patients are usually considerably higher than those operated on subsequently. It is indeed disturbing to contemplate that efficiency is gained at such a cost. Some comfort may be derived by the consideration of the fact that one is able to give relief by this method in cases that could not be dealt with in any other way.

Thirty-two patients have been operated on by this method; five died—an operative mortality of 15·6%. Of these five patients, three were very poor operative risks, two of them being over eighty and very feeble.

A Consideration of Statistics.

When one comes to consider the question of mortality rates associated with the operative treatment of prostatic enlargement, a great many factors have to be taken into consideration. Conclusions derived from the study of statistics can of course be delightfully or disappointingly misleading, and until some generally accepted standard of evaluating and considering every factor is brought into common use, that state of affairs will continue. I should think that a proper study of statistics would take into consideration, as well as the number dying after operation, the number who come under observation and die without operation. Frequently patients who are poor operative risks are admitted to hospital with retention of urine. After a period of catheter drainage they regain to a certain extent the power of micturition, owing to a subsidence of prostatic congestion. Because they are poor risks and because they are not actively seeking operation, they are often allowed to leave hospital. On the other hand, some surgeons might regard it as their duty to advise patients of this class that they were seriously ill, and that a long period of pre-operative treatment followed by the requisite surgical procedure was essential. Surgeons who fall into the latter class will almost certainly have a higher mortality rate than those who allow the question of statistics to influence their judgement. At the same time, in the long run they will probably save

more lives. Among other facts that should be considered in the evaluation of statistics are those of race, intelligence, diet, climate, dental equipment and associated other diseases.

However, taking all these points into consideration, a study of the figures does indicate that the mortality of prostatic surgery is still too high, especially in public hospitals. In 1933 the late F. W. Green,[17] in the *Royal Melbourne Hospital Clinical Reports*, showed a series of tables illustrating certain points regarding the mortality rate of 203 patients operated on at the Melbourne Hospital during the years 1927 to 1931 for benign enlargement of the prostate. Of these patients, 42 died, a mortality rate of 20·7%. He compares figures from twelve British hospitals and from Saint Peter's Hospital, which were 19·5% and 9·9% respectively. J. Newman Morris,[18] speaking at the Australasian Medical Congress held in Hobart in 1934, presented statistics from Saint Vincent's Hospital. He reported that in the twelve years from 1921 to 1932 some form of operative treatment was carried out on 127 men with prostatic obstruction. Of these, 33 died in hospital, a total mortality rate of 26%. B. T. Zwar[19] at the same congress reported that at the Melbourne Hospital during the eighteen months ending June 30, 1933, 152 patients suffering from symptoms of prostatic enlargement were admitted. Of these, 124 were subjected to operation and 31 died, a mortality rate of 25%. Of the 28 not operated on, 15 died within an average stay in hospital of three and a half days. In other words, 10% of the total number were apparently almost moribund on admission, which is a very unwholesome state of affairs. It would be reasonable to assume from these figures that another 20% to 30% would be very poor risks. When one compares with these results those obtained, for example, by the late S. H. Harris, who operated on over 700 patients with a mortality of under 3%, and also those obtained by several American surgeons operating by the suprapubic, perineal and transurethral routes, there are at least three conclusions one can draw without, I think, much fear of contradiction.

1. It is obvious that appropriate treatment in experienced hands, applied sufficiently early, results in low mortality figures.

2. Any method of treatment applied to the long-standing and neglected case will carry with it a considerable increase in the mortality rate.

3. There is too great a difference between the figures obtained in special hospitals and by individuals operators, such as Harris, and those obtained in the average general hospital.

I quite realize that at the present time the general hospital gets most of the "flotsam and jetsam" of prostatic obstruction. Surgeons are expected to operate on the type of patient who has been described as being possibly able "to stand a haircut and shave, provided that it was done very carefully in two stages".

At the same time I do feel that it should be possible to obtain eventually results in a general hospital that are at least comparable with those obtained in special hospitals and by individual operators, more especially, I think, if certain changes are made and special facilities are provided. Leaving out of the question surgical skill and experience, which are a *sine qua non* in any general hospital of standing, the important factors are beds, time, interest and, lastly, if I may use that rather hackneyed phrase, education, not only of the medical profession, but also of the general public.

During everyone's student days certain incidents occur and certain cases are encountered which undoubtedly have a definite influence on the individual's outlook in the future. I have endeavoured to throw my own mind back to my introduction to prostatic surgery. I can remember a sweating surgeon struggling in a sea of blood for what seemed an unconscionably long time, eventually removing a small piece of tissue, and I can remember the patient dying three days later. I can remember an incident in the casualty department, when I was instructed to catheterize an elderly gentleman. This being my first attempt at passing a catheter, I approached the patient, after carrying out my ablutions for the recommended ten minutes, with some trepidation, which was abundantly justified, for I found myself completely unable to do anything except cause him extreme discomfort. A passing dresser came to my assistance and, seizing the catheter, introduced it with the greatest of ease; urine gushed forth and the man departed on his way rejoicing. I was surprised to find later that he was suffering no obvious ill-effect from the laying on of the unwashed hands of the dresser.

Following that, my impressions were, firstly, that prostatic surgery was an extremely bloody and hazardous proceeding; secondly, that long-continued catheterization might be the lesser of two evils; and thirdly, that the necessity for this "scrubbing up" business seemed to be rather over-stressed. I think that numbers of students pass their final examinations without realizing that prostatic enlargement can cause an obstructive lesion which is almost as serious in its effects as a similar lesion occurring in the intestinal tract, and which calls for early efficient action.

I think that if it were possible to have patients with prostatic enlargement treated in a ward set apart for them, when time was not a factor, that a great many advantages would accrue. I shall endeavour to summarize these as I see them:

1. The pre-operative and post-operative nursing of these patients is of vital importance. It is reasonable to suppose that a sister given charge of such a ward should eventually develop into an expert in that most difficult branch of special nursing. Furthermore, the training of nurses in this particular work would be improved and simplified.

2. The training of resident medical officers and students would also be simpler and more efficient.

Cases seen *en masse* always make a more lasting impression. Students would see the type of patients who get well quickly, and would learn to realize the necessity for advising early operation when obstructive symptoms develop.

3. If all patients were nursed in one ward, the respective values of different methods of treatment would more easily and accurately be calculated.

4. It is easier and more economical to supply all that is necessary in the way of equipment to one such ward as opposed to a number of wards. If thought necessary, suction drainage could be laid on.

5. Such a ward should contain beds which are available for emergency cases when patients come to hospital requiring catheterization.

It would be argued probably that no hospital could afford this; but it also might be argued, and I think with greater truth, that no hospital can afford not to do it. Quite apart from the question of saving life, which can scarcely be calculated on a financial basis, it is very obvious that the more efficient the preparatory treatment, the shorter will be the time in hospital after operation. In my opinion there is only one place where a prostatic patient can be safely catheterized and adequately investigated and prepared for operation, and that is in bed in a hospital.

Regarding the question as to whether the surgery of the prostate in a general hospital should be in the hands of the many or a few, that is a matter that I do not feel qualified to discuss. I am certain, however, that the surgeon who spends much time in thinking about his patient before and after operation will in the long run obtain better results than he who is satisfied mainly with brilliant surgical technique.

In favour of my contention that it is attention to the apparently insignificant that counts in the surgery of the prostate, I will conclude by quoting some remarks by J. W. S. Laidley.[20] He states:

> In 1926 the death rate at the Royal Prince Alfred Hospital from suprapubic prostatectomy was in the region of 25%, and had stood about that figure for years. At that time the pre-operative and post-operative treatment was in the hands of several untrained wardsmen, who combined their prostatic duties with other work, such as carrying coal, transporting food and doing *post mortem* room work. On investigation it became evident that four out of every five deaths were due to pyelonephritis. The treatment of these patients was put in the hands of the female nursing staff for twelve months. There was no improvement. A second change was made and treatment was put in the hands of the resident staff for twelve months. Again there was no improvement. Finally a suitable individual from among the wardsmen was selected and given complete charge of the catheter-changing and irrigations. The result was immediate. The death rate fell to about 5% and has been in that vicinity ever since. . . . Here, then, is a fundamental and prime necessity for success: the concentration of patients under one responsible person, whose sole duty is the well-being of those patients.

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I desire to express my appreciation to my colleagues who have given me opportunities to do this work, and to Dr. Percy Smith, the superintendent of the hospital, and the sister in charge of the surgical ward.

References.

(1) O. S. Lowsley: "Development of Human Prostate Gland", *American Journal of Anatomy*, Volume XIII, 1912, page 290.
(2) Krogius: *Acta Chirurgica Scandinavica*, Volume LXXII, December, 1932, page 46.
(3) W. Boyd: "Surgical Pathology", 1929, page 516.
(4) Hugh Lett: *The British Journal of Surgery*, July, 1937, page 198.
(5) Frank Hinman: "The Principles and Practice of Urology", 1935, page 85.
(6) Kenneth Walker: "The Enlarged Prostate", 1926, page 25.
(7) *The British Journal of Urology, December*, 1933, pages 351-380.
(8) Alex. Randall: Quoted by F. Hinman in "The Principles and Practice of Urology", page 893.
(9) S. H. Harris: *The Australian and New Zealand Journal of Surgery*, January, 1935, page 231.
(10) J. Gray: *The British Journal of Urology*, September, 1932, pages 233-240.
(11) R. J. Silverton: *The Australian and New Zealand Journal of Surgery*, January, 1937, pages 305-306.
(12) S. H. Harris: *The Australian and New Zealand Journal of Surgery*, January, 1935, page 232.
(13) Frank Hinman: "The Principles and Practice of Urology", 1935, pages 332-333.
(14) Chevassu: *Gazette des hôpitaux civils et militaires*, 1933, page 106.
(15) Malcolm Baillie: *The British Journal of Urology*, March, 1935, page 33.
(16) N. C. Alcock: "Prostatic Hypertrophy", *The Journal of the American Medical Association*, March 2, 1935, page 734.
(17) F. W. Green: *Melbourne Hospital Clinical Reports*, June, 1933, page 60.
(18) J. Newman Morris: "Transactions of the Australasian Medical Congress (British Medical Association), Fourth Session, Hobart, 1934", page 346.
(19) B. T. Zwar: "Transactions of the Australasian Medical Congress (British Medical Association), Fourth Session, Hobart, 1934", pages 342-343.
(20) J. W. S. Laidley: *The Australian and New Zealand Journal of Surgery*, July, 1936, page 55.

THE DIAGNOSIS OF TUBERCULOUS DISEASES OF THE GLOBE.[1]

By J. Bruce Hamilton,
Hobart.

I am afraid I have chosen for today a rather difficult subject, but I feel it is one which should evoke some useful discussion.

I have frequently in past years asked myself the following questions: (*a*) What clinical or biological tests have we at our disposal for clinching a diagnosis of tuberculosis in diseases of the eyeball when for obvious reasons it is impossible to resort to biopsy? (*b*) What reliance can be placed on each of these tests in arriving at an accurate diagnosis?

When Dr. Geoffrey Black asked me to speak at this meeting on tuberculosis, I gladly accepted his invitation, and I have recently endeavoured while in Europe and America to ascertain the opinion of authorities on these two questions.

Although in the past I have been a constant adherent to the focal sepsis theory, yet events of the last three years have somewhat shaken my faith, and I am now in full accord with C. B. Goulden

[1] Based on a paper read at the fifth session of the Australasian Medical Congress (British Medical Association), Adelaide, 1937.

(1934), who recently stated that he was tending to revise the opinion he had held for many years, that tuberculosis of the eye was comparatively rare; his present view now was that it was more common than he formerly thought. Savin (1934) and Sorsby (1936) confirm this opinion. One factor that may be in my favour is this, that non-respiratory tuberculosis is more than twice as common in Tasmania as in other parts of Australia (Holmes, 1937). We may naturally take as a corollary to this that ocular tuberculosis is more prevalent in Tasmania than in the other States of the Commonwealth; but I have no figures to support this latter contention. Goulden nevertheless admitted that although he had been associated with a hospital for surgical tuberculosis for eighteen years in England, he had been struck by the apparent lack of active ocular tuberculosis in that hospital. Woods (1933) in America expresses a similar opinion, while W. D. Counsell (1937) in Tasmania informs me that during the past two years 236 new cases of pulmonary tuberculosis have been admitted to the Tasmanian Sanatorium, and to his knowledge not one active case of eye disease has occurred during that time in the institution. Despite these extraordinary facts, I still adhere to my previous statement that tuberculous disease of the eye is more common than most of us suspect, and I feel, as Edmund (1935) has shown, that it is most prone to occur in patients with healing or inactive chest lesions.

Today I have no intention of discussing those diseases of the orbit, lid, conjunctiva or lachrymal sac in which the diagnosis can be confirmed by pathological section, but to confine my attention to ocular diseases in which section is not feasible and in which the tubercle bacillus is sometimes (possibly frequently) the ætiological factor. I refer to the following diseases: (i) phlyctenular disease, (ii) interstitial keratitis and sclerosing keratitis, (iii) episcleritis and scleritis, (iv) iridocyclitis, (v) uveoparotitis, (vi) chorioiditis, (vii) retinitis, and (viii) vitreous hæmorrhage.

Having this list of diseases before us, let us survey our present-day means of arriving at a diagnosis and try to assess in the light of modern knowledge the value of each avenue.

Diagnostic Methods.

In the investigation of a possible case of tuberculous involvement of the eyeball the following avenues are generally explored: (i) family and environmental history, (ii) local clinical signs, (iii) diagnostic skin tests, (iv) X ray examination of the chest, (v) sputum, (vi) blood cultures, (vii) blood sedimentation rate and blood counts, (viii) other organs affected with tuberculosis, (ix) exclusion of other diseases, (x) response to treatment, and (xi) after-history.

I propose to deal with these methods *seriatim* and to discuss the intraocular diseases only when they illustrate a point or need special reference.

History.

I am of the opinion that a careful history of individual patients is of paramount importance, and this is confirmed by Derby and Carvill (1927). Contact with tuberculous friends or relatives can be found frequently in cases of phlyctens, iridocyclitis and retinitis. The infectivity of tuberculosis is unquestionable (Storey, 1936).

Phlyctenæ.—Sorsby (1936) has shown conclusively by his figures from Swanley Hospital that phlyctenular disease is a manifestation of tuberculosis of possible allergic nature. Woods (1933) and Bickerton (1935) are of a similar opinion, while Edmund (1935) has come to the same conclusion in observing the frequency of phlyctenular disease in pulmonary tuberculosis patients undergoing treatment with "Sanocrysin".

Recently a female patient, aged fifty years, suffering from bilateral clinical tuberculous iridocyclitis and exhibiting a very strong family history of tuberculosis, was given two courses of "Solganol B. Oleosum" by her physician at my request; during the second course of teratment, and while the cyclitis was subsiding, she developed a phlycten on her left eye.

Although phlyctens are rare in Tasmania, I have had fairly recently two patients with these diseases, the first a male aged seven years, with a phthisical mother, the second a female aged twenty-eight years, with a phthisical husband.

Iridocyclitis.—The only case of heterochromic iridocyclitis which I have seen in Tasmania was in an English woman married to a returned soldier who has a tuberculous spine. Further, my very latest patient with active advanced bilateral iridocyclitis with old chorioiditis in both fundi informed me that four of her husband's cousins died of phthisis and that one of these recently died in her home.

Retinitis.—Three years ago I had under treatment a young man of twenty-four, whose retinal lesions in both eyes were diagnosed as Eales's disease. After six months' treatment he completely recovered, with normal vision in both right and left eyes; and greatly to my surprise he has had no relapses. Two years later his younger sister reported with acute iritis in one eye, which rapidly resolved, to be followed one month later by acute retinitis and vitreous hæmorrhage of a similar nature to that of her brother. I then discovered that their mother, with whom they both lived, had had an open phthisical lesion for years.

More recently I saw in consultation a male, aged twenty-six years, with complete bilateral vitreous hæmorrhages of a recurrent nature, also diagnosed as Eales's disease. Careful inquiry elicited the fact that he lived with his sister, who had had an open chest lesion, now supposed to be inactive. I advised immediate removal of our patient from the focus of infection.

Of even more importance is the following history of a male, aged twenty-four years, complaining of spots before the eye. He had hæmorrhages and exudates along the veins in the periphery of both fundi, as described by Troncoso (1937). The ætiology was obscure until I discovered that he had shared a bedroom for months with a fellow student who was now dying of acute phthisis.

Local Clinical Signs.

It is at this point that I realize the difficulty of the situation, opinions differ so on both sides of

the Atlantic and also in the several capitals of Australia; but there are two features of tuberculosis of the eye which are familiar to all, and these are chronicity and almost constant tendency to recurrences (Derby and Carvill, 1927). The greatest help I have received is from Garland and Thompson (1933 and 1934) and I can refer you with confidence to their two articles. They have unmasked a syndrome of bilateral painless parotitis and bilateral uveitis frequently accompanied by transient facial palsy, and have conclusively proved by biopsy and autopsy and animal inoculation that the condition is undoubtedly tuberculous in origin. They confirm the opinion expressed by Savin (1932).

Finnoff (1933) has also described a syndrome of ocular tuberculosis with the following signs: (i) mutton-fat keratic precipitates, (ii) evanescent greyish nodules on the iris margin (Gilbert-Koeppe nodules), (iii) increased intraocular tension, (iv) vitreous exudates, and (v) one or more yellowish tubercles of the chorioid. He has not, however, been able to substantiate it by biopsy or animal inoculation.

In 1933 I saw a case of uniocular iridocyclitis with mutton-fat keratic precipitates and secondary glaucoma. Microscopic section of the eye showed typical gross giant-cell systems, but physical examination of the patient revealed no abnormality. Keratic precipitates then appeared in the other eye, so tuberculin therapy was commenced; but within three months the patient had died of fulminating generalized miliary tuberculosis.

I think at least we may accept mutton-fat keratic precipitates as fairly pathognomonic of tuberculous iridocyclitis.

Attention must also be called to the evanescent greyish nodules on the iris margin (Gilbert-Koeppe nodules). I saw these once in a patient with an active chest lesion, and was surprised how rapidly they disappeared on the exhibition of atropine and heat. Both Woods (1933) and Finnoff (1933) consider that they are a manifestation of tuberculous iridocyclitis. Long-standing tuberculous nodules of the iris I have not seen in Tasmania, but Thiel (1937) gives perfect illustrations of such a condition in his atlas.

Another interesting condition of the iris, and one supposed to indicate a tuberculous ætiology, is heterochromia. As I indicated in my opening remarks, my only patient with this condition has a family history of tuberculosis.

I do not think I need mention phlyctenular keratitis, as everyone is conversant with its clinical signs. All I need do is again to refer you to Sorsby (1936) in *The Transactions of the Ophthalmological Society*.

Of course, conglomerate tubercles and multiple miliary tubercles in the fundus have been accepted almost universally, but are rare conditions, especially in Australia. I wonder how many present today have seen five cases of conglomerate tubercle and how many have seen multiple miliary tubercles of the retina, except in the last stages of a fulminating generalized miliary tuberculosis?

This brings me to three conditions of the fundi not so rare, all of which I consider are tuberculous. Firstly, there is that perivasculitis so ably described and pictured by Troncoso (1937), which was exhibited by my three patients described under the heading "Family and Environmental History". This perivasculitis is frequently accompanied by massive vitreous hæmorrhages and is then commonly designated Eales's disease. Williamson-Noble (1936) reports Gilbert as having isolated tubercle bacilli in the perivascular sheath of a retinal vein of an eye excised for perivasculitis.

Secondly, there is a low-grade disseminated chorioiditis of a very diffuse but mild nature. Each patch has a creamy and only slightly raised centre with faintly pigmented border, the whole appearing like the ghost of an active disseminated chorioiditis. The end result is a retina with multiple patches of incomplete atrophy, each patch having a pigmented edge and the whole retina finally presenting a moth-eaten appearance. W. D. Counsell will shortly report such a typical picture of tuberculous retinitis, in which case an enlarged epitrochlear gland was excised and sectioned and was reported to exhibit tuberculous lymphadenitis. In Counsell's case the retinitis was accompanied by mutton-fat keratic precipitates.

The third condition I mention is that of isolated patches of old chorioiditis, so often found on routine examination. These patches are characterized by a large white plaque of atrophy, surrounded by a thin edge of aggregated pigment. Troncoso (1937) describes these patches as absolutely indicative of a tuberculous agency and, speaking of the lack of pigment, says: ". . . chorioidal tuberculosis destroys pigment, whereas syphilis fabricates it." Aruga (1936) considers that old equatorial and peripheral chorio-retinitis found before or at the time of retinal detachment in adults is in most cases tuberculous in origin. He also considers that juvenile detachments—unilateral or bilateral, and inferiorly situated—constitute a special group and are probably tuberculous in origin also. I have seen only three cases of juvenile detachment that would come under this heading, and these I reported in my recent Gifford Edmonds Essay (1937). I found no evidence in these three cases to support or disprove this theory. May not the precursor of these patches of old chorioiditis be the yellowish tubercles of the chorioid described by Finnoff (1933)?

Adler and Myer (1937) have pointed to the fact that tuberculous lesions of the eye vary with age. In persons up to twenty years of age they are of the nodular type, for example, phlyctens, nodular iritis, miliary tubercles in the chorioid; from twenty to thirty years of age they are of the inflammatory type, for example plastic iritis and retinal periphlebitis; and from thirty to fifty years of age they are of an exudative nature, for example chronic iridocyclitis with mutton-fat keratic precipitates and disseminated chorioiditis. These workers also point out that allergy to tuberculo-protein increases with age.

Skin Tests.

The two skin tests most commonly used in the diagnosis of tuberculosis are the von Pirquet cutaneous test and the Mantoux graduated intradermal test, and I have made a point of inquiring from clinicians of authority, both in Europe and America, into the reliability of these tests. It appears to be universally acknowledged that a positive response to the von Pirquet test in any individual over five years of age is absolutely useless; Woods (1933) and Garland and Thompson (1933) show very conclusively that a negative result in adults is equally unreliable. So it appears that the von Pirquet test should at once disappear from our diagnostic armamentarium, except in children under five years of age. This leaves us with the Mantoux test, faith in which appears to vary on both sides of the Atlantic.

Urbanck (1932) and Meller (1934), both of Vienna, report favourably on graduated intracutaneous tuberculin tests with "Tebeprotin", according to the technique of Mantoux, and stress the importance of avoiding a focal reaction. Some clinicians, on the other hand, consider a positive response diagnostic only when a "++++" reaction results. Finnoff (1928) places no reliance in a positive local reaction, but considers a focal reaction in the eye of greatest significance, while admitting the danger. Williamson-Noble (1936) asserts that a positive Mantoux reaction in an adult has very little diagnostic significance. Goldenburg (1935), speaking of tuberculin in ophthalmology, says:

> This writer discontinued the use of tuberculin, both for diagnostic and therapeutic purposes, about fifteen years ago, and has not encountered any evidence, written or by clinical contact, sufficiently impressive to warrant a return to such management.

Poncher (1935) says:

> A positive tuberculin reaction in general merely indicates a previous infection with tubercle bacilli, and this may occur in clinically non-tuberculous individuals.

He further points out that a failure to react to the tuberculin test may occur in toxic tuberculous states and in disseminating miliary first infections, after exposure to ultra-violet light, during pregnancy, in old age, and during the course of tuberculin therapy. I think his statements fairly sum up the position, and they are confirmed by Friedenwald and Dessoff (1935).

One must immediately ask, then, what significance have Sorsby's (1936) high rate of positive results in phlyctenular children and low rate in children with blepharitis at Swanley. Woods (1933) points out that various experimenters have shown that phlyctenular children are not only hypersensitive to tuberculin, but also to horse serum when injected intradermally. A further series of patients should be tested against tuberculin in one arm and horse serum in the other to confirm this assumption. I hope to carry this out and to report my findings at a later date.

I feel at present that too much credence should not be given to the Mantoux test, especially when a positive reaction is obtained, and that until we understand a great deal more about allergy in tuberculous infections we should cease placing much reliance on the significance of these skin reactions. One has but to recall the fact that 90% of the population are infected by tuberculosis at one or other period of their lives, scarred chest lesions being found at autopsy, to understand the doubtful value of diagnostic skin reactions. I need hardly say that the conjunctival test of Calmette is no longer justified, and that the serious reactions in the eye caused by its employment must necessitate its abandonment.

X Ray Examination of the Chest.

As the lungs are the commonest site of tuberculous infection, attention naturally turns to the chest in all suspected lesions of the eye, and the physician to whom the patient is referred for an opinion is frequently asked to concentrate on that aspect. The report usually received may fall into one of three categories: (i) "negative" chest findings, (ii) evidence of old chest lesions, (iii) active chest lesions. Seldom is an expression on the relation of the chest signs to the eye lesion obtained.

While in London I was able to discuss this aspect of the subject with W. D. Wykeham Brooks, of Saint Mary's Hospital, and he has given me permission to quote him, although his findings will not be published until later in *The British Journal of Ophthalmology*. Working with F. A. Juler, of the ophthalmic department of the same hospital, on patients with iridocyclitis and uveo-parotid fever, he has found almost consistently enlarged hilar glands in the chest, resembling very closely those found in Hodgkin's disease, and of such typical character that he considers them almost pathognomonic of tuberculous uveitis.

Sputum Examination.

The question of sputum examination is again usually the prerogative of the physician, but naturally it will not be performed by him unless the oculist indicates that the eye is possibly tuberculous.

Repeated sputum examinations revealing no tubercle bacilli are not considered sufficient by Ulmar and Ornstein (1935). In 287 cases, tubercle bacilli repeatedly failing to appear in the sputum, they performed gastric lavage; tubercle bacilli were obtained from the gastric contents in approximately 25% of cases. Holmes (1935) stresses the advantages of attempting culture from the sputum in such cases. Procedures such as gastric lavage may on the surface appear a little drastic, but when a differential diagnosis between phthisis and carcinoma of the lung is essential, the usefulness of this new technique becomes very apparent. These clinicians are emphatic that "no examination for tubercle bacilli is complete unless there has finally

been a gastric lavage and examination of the stomach contents for tubercle bacilli".

Blood Cultures.

Meller (1934), in his Doyne Memorial Lecture, appears impressed with the work of E. Löwenstein, who obtained 14% positive blood cultures from patients with supposedly tuberculous eye lesions. Mellon (1935) views Löwenstein's work with some scepticism, but admits that the obscure biological behaviour of the tubercle bacillus, and especially its transference from non-acid-fast to acid-fast forms, makes it difficult to assess the value of Löwenstein's work. Meller, in the concluding remark of his lecture, begs for a continuation of Löwenstein's work in other clinics, and also for tissue cultures from all suspected specimens sent for microscopic examination.

Blood Sedimentation Tests and Blood Counts.

Lyle Cummins (1934) discussed the value of blood sedimentation tests, of the Arneth count, of the lymphocyte-monocyte ratio, and of the total leucocyte counts in tuberculosis, and was of the opinion that these tests were of little or no value by themselves in diagnosis of tuberculous lesions, but were valuable in assessing the constitutional state of the patient from time to time during the course of treatment. Beaumont and Dodds (1936), speaking of blood sedimentation tests alone, are of the same opinion, while Koch and Mellon (1935) say that a normal sedimentation rate renders the presence of an active tuberculous process very improbable.

I think that, like the diagnostic skin reaction, these tests should not weigh too materially in our diagnosis of tuberculosis.

Other Organs Affected with Tuberculosis.

Derby and Carvill (1927) found amongst 63 patients with ocular tuberculosis (phlyctenular keratitis, sclero-keratitis, uveitis and tuberculous keratitis), that 19 had lung involvement, 21 had adenitis, 3 had pleurisy, 3 had tuberculous mediastinal glands, and 2 had tuberculous synovitis, and that 9 other patients showed evidence of tuberculosis in sites other than the globe. This point will be further elaborated under the section devoted to after-history.

Exclusion of Other Diseases.

It is surprising in consultative work how often the Wassermann test, urine examination, blood counts and other biochemical tests have been omitted. One has only to recall the similarity between syphilitic and tuberculous lesions of the eye, or the relation of glycosuria to human tuberculosis, to realize the imperative necessity of complete biochemical examinations. Some clinicians excuse themselves by emphasizing the economic factor, but I find that a full primary examination frequently saves the patient both unnecessary treatment and unnecessary suffering.

Result of Treatment.

When there is so much apparent divergence of opinion as to the results of tuberculin therapy, it is difficult to lay any stress on the result of treatment with this vaccine [Wilkinson (1933), Goldenburg (1935), Derby and Carvill (1927), Flint (1934)].

Recently I have been treating my patients with chronic uveitis of possible tuberculous ætiology with "Solganol B. Oleosum", and I considered the results most satisfactory. This is confirmed by Benedict and Goecherman (1932). I was surprised to find that authorities such as Kayne (1935) and Beaumont and Dodds (1936) are very doubtful as to the efficacy of gold salts in pulmonary tuberculosis, but many authors, including Parr and Shipton (1937), find them of great assistance in rheumatoid arthritis.

With such conflicting opinions of specific and empirical therapy in tuberculosis, I feel we cannot at present confirm a previous diagnosis of tuberculous disease of the globe in the light of response to treatment.

After-History.

Here we have a definitely helpful sphere, and one which should be utilized universally.

Savin (1932) points out when discussing the prognosis of uveoparotitis that every patient who died from the disease showed evidence of miliary tuberculosis at autopsy; while Sorsby (1936), in a follow-up of 238 children with phlyctenular diseases, found that 14 developed active tuberculous lesions in other parts of the body. Derby and Carvill (1927) report 33 cases of phlyctenular keratitis, six patients dying of phthisis.

I feel certain that if all our patients with presumed tuberculosis of the eye could be followed to death, a surprising proportion would be found to have developed tuberculous lesions in other parts of the body, and not a few would be found to have died from generalized tuberculosis.

A follow-up system of all patients with clinically tuberculous eye disease should be both enlightening and instructive, and might possibly clear away much of the thick haze which now envelops the subject under discussion today.

Conclusion.

I should like to draw your attention to the frequency with which I have referred to the work of A. C. Woods (1933) in this paper and to urge the reading of his monograph on the subject of allergy and immunity in ophthalmology. You may not agree with many of his conclusions, but he will at least bring to your view a new aspect of the ætiology of many ophthalmic diseases. Here we find interesting light thrown on the relation of allergy to immunity, of allergy to tuberculin diagnostic skin reactions, of allergy to phlyctenular disease, to keratitis and to sympathetic ophthalmia.

You will no doubt find it hard to correlate Woods's findings in sympathetic ophthalmia with those of Meller (1934), but so far as I could ascertain in America, Woods's work on uveal pigment testing was accepted in some clinics; and I think you will agree that he has at least quoted a vast field of experiments to substantiate his opinions. One experiment by Finnoff, quoted by him, namely, the production of typical human tuberculous eye lesions in rabbits by injecting them with dead tubercle bacilli, conclusively demonstrates the fact that allergy is undoubtedly an all-important factor in ocular tuberculosis, and one demanding our constant attention if we are to try to arrive at a more accurate diagnosis in future.

Bibliography.

F. H. Adler and G. P. Meyer: *Archives of Ophthalmology*, Volume XVIII, Number 2, 1937, page 275.

H. Arruga: "Detachment of the Retina", 1936.

G. E. Beaumont and E. C. Dodds: "Recent Advances in Medicine", 1936.

W. L. Benedict and W. H. Goecherman: *Archives of Ophthalmology*, Volume VIII, 1932, page 250.

C. Berens: "The Eye and its Diseases", 1936.

R. E. Bickerton: *Tubercle*, Volume XVII, 1935-1936, page 193.

W. D. W. Brooks: Personal communication, London, 1937.

W. D. Counsell: Personal communication, Hobart, 1937.

S. L. Cummins: *The General Practitioner*, February 15, 1934, page 379.

G. S. Derby and M. Carvill: *Archives of Ophthalmology*, Volume LVI, 1927, page 523.

C. Edmund: "Clinical Studies of Endogenous Conjunctival Affections", 1935.

W. C. Finnoff: *Journal of the American Medical Association*, Volume XC, 1928, page 341.

W. C. Finnoff: *Archives of Ophthalmology*, Volume IX, 1933, page 13.

G. B. Flint: *Edinburgh Medical Journal*, Volume XLI, 1934, page 235.

J. S. Freedenwald and J. Dessoff: *Bulletin of the Johns Hopkins Hospital*, Volume LVI, 1935, page 148.

H. G. Garland and J. C. Thompson: *The Lancet*, Volume II, 1934, page 743.

M. Goldenburg: "Goldberg's Clinical Tuberculosis", Volume II, 1935.

C. B. Goulden: *Transactions of the Ophthalmological Society of the United Kingdom*, Volume LIV, 1934, page 95.

C. B. Goulden: *Transactions of the Ophthalmological Society of the United Kingdom*, Volume LVI, 1936, page 80.

J. B. Hamilton: Gifford Edmonds Prize Essay in Ophthalmology, 1937.

E. M. Holmes: *Proceedings of the Royal Society of Medicine*, Volume XXVII, Number 4, 1935, page 43.

M. J. Holmes: THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, Number 19, 1937, page 813.

C. G. Kayne: *Proceedings of the Royal Society of Medicine*, Volume XXVIII, 1935, page 1463.

R. R. Koch and R. R. Mellon: "Goldberg's Clinical Tuberculosis", Volume I, 1935.

J. Meller: *Transactions of the Ophthalmological Society of the United Kingdom*, Volume LIV, 1930, page 467.

R. R. Mellon: "Goldberg's Clinical Tuberculosis", Volume I, 1935.

C. G. Ornstein and D. Ulmar: *Ibidem*.

L. J. A. Parr and E. C. Shipton: THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, June 5, 1937, page 864.

H. G. Poncher: "Goldberg's Clinical Tuberculosis", Volume II, 1935.

L. H. Savin: *Transactions of the Ophthalmological Society of the United Kingdom*, Volume LVI, 1934, page 549.

A. H. H. Sinclair: *Edinburgh Medical Journal*, Volume XLI, 1934, page 233.

A. Sorsby, H. Hamburger and L. R. Benham: *Transactions of the Ophthalmological Society of the United Kingdom*, Volume LVI, 1936, page 63.

J. C. Storey: THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, Number 20, 1936, page 670.

R. Thiel: *"Atlas der Augenkrankheiten"*, 1937.

U. T. Troncoso: "Internal Diseases of the Eye", 1937.

D. J. Urbanck: *Transactions of Ophthalmological Society of the United Kingdom*, Volume LII, 1932, page 227.

W. Wilkinson: "Tuberculin: Its Vindication by Technique", 1933.

F. A. Williamson-Noble: *The British Medical Journal*, November 7, 1936, page 907.

A. C. Woods: "Allergy and Immunity in Ophthalmology", 1933.

THE TREATMENT OF CLONIC FACIAL SPASM BY NERVE ANASTOMOSIS.

By GILBERT PHILLIPS, M.S., M.Sc.,

Honorary Neurosurgeon, Lewisham Hospital; Assistant Honorary Neurosurgeon, Royal Prince Alfred Hospital.

(From the Department of Surgery, The University of Sydney.)

TRUE clonic facial spasm is a unilateral, intermittent or clonic series of contractions of the facial muscles limited to one side. Its pathology is not known. It has been suggested to be a degenerative neuritis of the facial nerve in the region of the geniculate ganglion. It is commoner in females than in males. It is slow in onset and first appears as an intermittent blepharospasm on one side. Later the remaining facial musculature on the same side becomes involved, and in severe cases there are clonic contractions also of the platysma and occasionally of the stapedius muscles. When fully developed, the condition may thus involve the whole of the musculature supplied by the facial nerve on one side in a persistent series of clonic contractions, which are most annoying and frequently debilitating to the patient, and ultimately result in contractures or paralysis of the muscles involved. During sleep the spasms cease.

This condition may be cured by replacement of the innervation of the muscles involved by some motor nerve other than the facial. The nerves which are most frequently used are the accessory, hypoglossal or the *descendens hypoglossi*. The method of nerve anastomosis in the treatment of clonic facial spasm is relatively recent, and in an article in 1932[1] Harris and Wright stated that they could find only two previous examples of such an operation in the literature.

In deciding whether to use the accessory or the hypoglossal nerve as the source of central innervation the occupational factor must be considered. In labourers or in those who habitually use the shoulder to support weights, the hypoglossal nerve should be employed, since considerable weakness of the trapezius results from section of the accessory nerve. If the hypoglossal nerve is used, hemiatrophy of the tongue takes place, but in most cases this produces little or no disturbance in speaking. In persons of sedentary occupation the accessory nerve should be used. At operation the facial nerve is sectioned at the stylomastoid foramen and its peripheral end is sutured to a central end, either of the hypoglossal or the accessory nerves. The immediate facial palsy which occurs is not so obvious as that resulting from other causes owing to the existence

of more or less contracture in the facial muscles. Regeneration takes place in three to four months. Movement is first seen in the lower facial muscles

FIGURE I.
Involuntary clonic facial spasm.

and appears primarily, if the hypoglossal nerve is used, when the tongue is pushed forwards, and if

FIGURE II.
Appearance of the patient after section of the left facial nerve and primary suture of its peripheral end to the central end of the left accessory nerve. Facial palsy is not particularly obvious, owing to the presence of some contracture in the facial muscles.

the accessory nerve is used when the shoulder is elevated. Within two or three weeks of the first appearance of movement in the lower part of the face reinnervation of the *orbicularis oculi* is completed. If the hypoglossal nerve is used, reinnervation of the frontalis muscles does not take place.

FIGURE III.
The patient wearing a Jennings splint.

Reeducation in front of a mirror soon enables the patient to perform voluntary movements on the affected side, but in a number of cases the movement

FIGURE IV.
Voluntary movement of the lower facial musculature on the left side three and a half months after operation. At this early stage this movement is produced by elevating the left shoulder.

remains diffuse and all facial muscles on that side tend to function in unison. Emotional expression on the same side is considerably impaired, and although by reeducation emotional movements may

be imitated, there is always an obvious impairment of spontaneity. During the regeneration period massage and galvanic stimulation are desirable, and a Jennings splint (see Figure III) may be used to support the angle of the mouth and to prevent "ballooning" of the cheek. In skilled hands the likelihood of regeneration not taking place is negligible. The patients are universally delighted with the results of surgical treatment. The following history illustrates a typical result, and the photographs accompanying this article demonstrate the debilitating nature of the condition in a patient engaged in active commercial life, the appearance of the face immediately after section of the facial nerve and primary suture of its peripheral end to the central end of the accessory, the method of using the Jennings splint, and the appearance after regeneration has begun.

Clinical History.

The patient, L.M., a young man aged twenty-seven years, is a wholesale dressmaker employing a large number of female assistants. The present condition had appeared about nine years ago as an intermittent twitching of the left *orbicularis oculi* muscle. The twitching became more frequent and commenced to involve the left side of the mouth and the left cheek. For the past two years paroxysms of twitching lasting several minutes had taken place seven or eight times an hour. These paroxysms were more prolonged and more severe when he was tired or worried. The platysma was not involved and there were no paroxysmal clicking noises in the left ear suggesting involvement of the stapedius. On complete neurological examination no abnormal physical signs could be detected, with the exception of clonic facial spasm. The other possible causes of clonic facial spasm were eliminated (local injury, cerebello-pontine angle tumour, encephalitis, post Bell's palsy).

At operation on June 4, 1937, an incision two inches long was made parallel with the anterior border of the mastoid process downwards and forwards to the angle of the mandible. The external jugular vein was retracted backwards and the posterior belly of the digastric muscle was identified. The accessory nerve was found passing downwards and backwards, after emerging under the posterior belly of the digastric, across the transverse process of the atlas. The facial nerve was identified by the branch which it gives to the upper border of the posterior belly of the digastric. The facial nerve was sectioned at the stylomastoid foramen and its peripheral end was swung downwards and forwards. The accessory nerve was sectioned as it passed under the anterior belly of the sternomastoid muscle and the central end swung upwards and passed medial to the posterior belly of the digastric. The two cut ends were then sutured with finest gauge Pearsall's silk. The point of union of the two nerves was then placed on the medial side of the posterior belly of the digastric muscle to isolate it from the operation field and thus to prevent involvement of the regenerating nerve fibres in fibrous tissue. The skin edges were approximated with Michel clips. Convalescence was uneventful except for transient clicking noises in the ear, due to reflex irritation of the stapedius. The patient was discharged from hospital five days after operation. The wound was quite inconspicuous. Three and a half months after operation he reported reappearance of movement at the angle of the mouth on the left side on elevating the left shoulder.

Reference.

(1) Wilfred Harris and A. D. Wright: "Treatment of Clonic Facial Spasm", *The Lancet*, Volume I, March 26, 1932, page 657.

Reports of Cases.

PROFOUND ANÆMIA FOLLOWING ADMINISTRATION OF SULPHANILAMIDE IN ACUTE GONORRHŒA.

By Edna L. Nelson, M.B., Ch.M.,
Director of the Special Clinic,

and

Margery Scott-Young, M.B., B.S.,
Medical Superintendent of the Rachel Forster Hospital for Women and Children.

Clinical History.

The patient, Mrs. E. J., aged thirty-two years, first attended the venereal diseases clinic of the Rachel Forster Hospital on August 31, 1937. On a previous visit to the out-patient department smears from urethra and cervix had been taken and found positive for gonococci.

The history and physical findings on August 31, 1937, were as follows:

On August 31, 1937, there was a history of vaginal discharge for three weeks, with the onset after a menstrual period; severe abdominal pain occurred for one day before the first visit (August 18, 1937), with anorexia and nausea, but no urinary symptoms. She had severe lower abdominal pain and vomiting.

There was nothing of importance in the previous history. The patient had been married for nine years; she had one child, aged six years, and had had no miscarriages. Her husband had been attending a clinic for treatment of gonorrhœa for one month. The patient's menstruation was regular; her period was due in two days.

Physical examination revealed a good general condition. The patient was pale and had generalized tenderness in both iliac fossæ. Pus was present in the urethra and paraurethral glands. The orifice of the right Bartholin's gland was reddened; pus was present. Cervicitis was present with mucopus, but no erosion. The fornices were tender when the uterus was moved; fullness was present in the left fornix. Mucopus was present in the rectum.

As recent reports from American workers[1] seem to indicate the usefulness of sulphanilamide in the treatment of gonococcal infections, it was decided to test the efficacy of the drug in this obviously acute case, and to use no local treatment whatever. In view, however, of a report in *The British Medical Journal* of July 17, 1937,[2] of a case of agranulocytosis following the use of sulphanilamide, it was decided to keep a check on the blood picture, particularly the leucocytes, during administration of the drug. The drug to be used was sulphanilamide (Parke, Davis and Company) five-grain tablets.

A preliminary blood count on August 31, 1937, gave the following results:

Hæmoglobin value	75%
White cells, per cubic millimetre	10,650
Polymorphonuclear cells	78%
Lymphocytes	17%
Monocytes	4%
Eosinophile cells	1%

No anisocytosis, poikilocytosis or nucleated red cells were seen and the staining reaction was normal.

A dosage of 50 grains of sulphanilamide in twenty-four hours was instituted on August 31, 1937, to be continued for three days. After taking 30 grains the patient had four loose bowel actions and thereafter no similar reaction. She was admitted to the ward on September 1, 1937. On the second day of treatment the patient was free of abdominal pain and vomiting. The urogenital signs remained unchanged, but there was slight tenderness only in fornices. Blood counts made on September 1, 1937, and September 2, 1937, revealed no change.

On the third evening of treatment the patient complained of severe headache, and later she became drowsy and slightly cyanosed. The dosage of sulphanilamide was reduced to 35 grains in twenty-four hours.

The report on blood taken on September 3, 1937, was as follows:

Red cells, per cubic millimetre	4,140,000
Hæmoglobin value	60%
White cells, per cubic millimetre	12,200
Polymorphonuclear cells	67%
Lymphocytes	28%
Monocytes	1%

Anisocytosis was present. There was no poikilocytosis. No nucleated red cells were seen. An occasional cell showed punctate basophilia.

Smears taken from all infected areas were reported "negative" for gonococci. The patient's general condition improved and no further effects of drug treatment were noted. Dosage was maintained at 35 grains in twenty-four hours.

Blood counts taken daily from then on remained at the level of September 3, 1937, with no abnormality of red cells.

On September 7, 1937, the patient was discharged from the ward, to report daily at the clinic. From September 8, 1937 to September 15, 1937, the patient's general condition was satisfactory and all clinical signs of gonorrhœa were absent.

On September 17, 1937, the dosage of sulphanilamide was reduced to 20 grains a day. The patient failed to report until September 22, 1937, when she returned complaining of malaise, anorexia and vomiting of two days' duration.

On September 22, 1937, the patient appeared ill, was cyanosed and her conjunctivæ were icteric. There was no clinical sign of gonorrhœa. A blood count yielded the following information:

Red cells, per cubic millimetre	2,970,000
Hæmoglobin value	43%
Colour index	0·74
White cells, per cubic millimetre	10,050
Polymorphonuclear cells	68%
Lymphocytes	24%
Eosinophile cells	8%

Anisocytosis was present and occasional microcytes were seen. Poikilocytosis was absent. Basophilia was present. There was a marked deficiency in hæmoglobin content.

From the general appearance of the patient, the rapid fall in red cell count and microscopic appearance of red blood cells, it was considered obvious that the patient's condition was similar to that of cases reported by Harvey and Janeway[3] as acute hæmolytic anæmia.

No further pathological tests were made, as the diagnosis did not appear to be in doubt. The patient was admitted to the ward. At this time the red cell count had fallen to 2,180,000 per cubic millimetre, the hæmoglobin value was 38%, and the colour index was 0·9.

A transfusion of 450 cubic centimetres of citrated blood was given into the forearm. Liver extract was given intravenously and four cubic centimetres intramuscularly. The red cell count next day was 3,680,000 per cubic millimetre, the hæmoglobin value was 48%, and the colour index was 0·66. The patient's general condition was much improved.

Two further transfusions of 450 cubic centimetres of citrated blood were given as well as daily intramuscular injections of liver extract.

On October 8, 1937, the red cell count had risen to 4,450,000 per cubic millimetre, the hæmoglobin value was 65%, the colour index was 0·73, and there was no abnormality of red cells. All smears taken from infected areas remained negative for gonococci.

On October 11, 1937, the patient was discharged from the ward. Since her discharge four blood counts have been made, and all made to date (November 5, 1937) show steady improvement. Her general clinical condition is excellent, she is clinically free from gonorrhœa, and all smear tests are "negative" for gonococci.

Commentary.

The following points may be noted:

1. The efficacy of sulphanilamide in the treatment of acute gonorrhœa. In spite of the complete absence of local treatment, all smears became "negative" for gonococci after two days, an incipient salpingitis was aborted, and the patient at the end of three weeks was completely clear from a clinical point of view.

2. The possibility of blood dyscrasia in the use of sulphanilamide. It may be remarked that the white cell count was the one upon which closest check was kept during the administration of the drug in this particular case. This was done because the possibility of agranulocytosis was uppermost in the minds of those in charge of the patient. The possibility of a hæmolytic anæmia had not been seriously considered, the report of the first cases of this complication having come to hand only recently.[3]

3. The necessity for hospitalization and close supervision when sulphanilamide is being given in large doses. Sufficient cases of hæmolytic anæmia have been reported to date to make one justifiably suspicious that they are not merely coincidental. It would seem a necessary, precaution, then, when administering large doses of sulphanilamide, to keep the patient under observation and to check the blood picture by frequent red and white cell counts.

Acknowledgements.

Our thanks are due to Dr. Addie Walker for her interest in and agreement to publishing notes of the case. We also wish to acknowledge the supply of sulphanilamide for clinical trial from Parke, Davis and Company, Sydney.

References.

[1] J. E. Dees and J. A. C. Colston: "The Use of Sulfanilamide in Gonococcic Infections: Preliminary Report", *The Journal of the American Medical Association*, May 29, 1937, page 1855.
[2] C. J. Young: "Agranulocytosis and Para-Amino Benzene Sulphonamide", *The British Medical Journal*, July 17, 1937.
[3] A. M. Harvey and C. A. Janeway: "The Development of Acute Hæmolytic Anæmia during the Administration of Sulfanilamide (para-aminobenzene-sulfonamide)", *The Journal of the American Medical Association*, July 3, 1937, page 12.

Reviews.

MODERN DIETARY TREATMENT.

A VALUABLE addition to the literature on the modern principles and application of nutrition is found in "Modern Dietary Treatment", by M. Abrahams and E. M. Widdowson.[1]

Miss Widdowson is already well known to many interested in dietetics for her work in association with McCance on nutritive values, carried out and published under the auspices of the Medical Research Council.

Miss Abrahams and Miss Widdowson have given in this book a clear and practical survey of dietary knowledge, as it stands today, together with a number of very valuable tables and diets for use in the treatment of various pathological conditions.

The first section deals with the principles of dietetics, and up-to-date knowledge is set out in a clear and simple way. The second section deals with the different diseases in which dietary treatment is necessary, and explains the reasons for the type of food employed. The third section gives detailed examples of diets for use in such diseases, and is particularly useful because of the variety of dishes provided.

[1] "Modern Dietary Treatment", by M. Abrahams, M.A., M.Sc., and E. M. Widdowson, B.Sc., Ph.D.; 1937. London: Baillière, Tindall and Cox. Crown 8vo, pp. 336. Price: 8s. 6d. net.

Especially valuable should prove the excellent tables, which are arranged to facilitate the planning of accurate yet varied diets. A number of recipes are included, which have been designed with the particular aim in view of suiting the several different types of diet.

Careful study of the book shows that it is scientifically sound and incorporates the most up-to-date ideas on dietary treatment. Recent information on the composition and nutritive value of foods has been incorporated in both the tables and the diets, and the book should prove one of the utmost value to doctors, nurses and others interested in dietetics.

ABNORMALITIES OF THE EYE.

Miss Ida Mann's latest work, "Developmental Abnormalities of the Eye", is an excellent volume.[1] We are now so accustomed to a high standard from her pen, and to original work, that this volume comes as a worthy companion to "The Development of the Human Eye". It is an inspiration to read her volumes, and as a reference they stand as somewhat monumental in English ophthalmic literature. The Cambridge University Press, too, has made splendid work of the publication—attractive cover, excellent paper, and print of the best standard.

The subject matter occupies some 421 pages and is of the greatest value to the clinical ophthalmologist, and to the man who takes so much interest in his specialty as to wish to know just why certain of his findings are there, and how their presence may be explained. The only criticism we have to offer is that there is no index to the 284 figures which illustrate the text; these include more than 60 coloured illustrations, all of which call for expression of admiration at the technician's art, both of drawing and reproduction.

That the ophthalmologist needs to keep his reading up to date might be emphasized by quoting the context of the Laurence-Moon-Biedl syndrome: "The condition seems to be an autosomal recessive and to involve linkage, the defects being of an epiblastic and a mesoblastic gene on the same chromosome."

There is so much in the volume that is readable, easily digested and informative as to make it an essential for the reference shelf, and as an authority it will have to be regarded as the last word on the subject.

EMANOTHERAPY.

"Emanotherapy", by Humphris and Williams, although dealing with the emanations of radium and certain allied substances, does not contain any reference whatsoever to the very important rôle of radium emanation in the treatment of neoplasms.[2] Its subject matter is confined to the use, and one is almost tempted to say abuse, of radioactive waters, both natural and artificially prepared.

There is no doubt about the authors' enthusiasm for their work, amounting to a degree which perhaps clouds their critical faculties. For example, the opening sentence, "It has been proved that the curative value of these and all similar waters is due to the emanations of radium that they contain", does not accord with the observation of Professor Russ, that the waters of a certain famous Swedish spa are less radio-active than the reticulated water supply of a neighbouring town. Nor are their claims for the value of this treatment in most of the diseases to which flesh is heir confirmed by recent experiments with artificially prepared radio-active water at Saint Bartholomew's Hospital. The latter experiments, in which both in-patients and out-patients with various forms of rheumatic trouble were used, did not give rise to any improvement other than some possible improvement in gouty subjects. That spa treatment will show good results cannot be doubted, but the authors' assumption that all such results depend only upon the radio-activity of the waters has directed them along debatable paths.

The most interesting part of the monograph is that dealing with radium poisoning. It is pointed out that all recorded instances of poisoning with radium have been due to the entrance into the body of a radium salt, while radon, or radium emanation, is quickly excreted, mostly by the lungs, whether it reached the body by inhalation, ingestion, inunction, insufflation, douches or other methods. It seems that a combination of these methods is desirable in the treatment of any given area of the body. Perhaps the rapid excretion of radon is a fortunate occurrence.

ENDOCRINES IN OBSTETRICS AND GYNÆCOLOGY.

Raphael Kurzrok, of Columbia University, has produced a book which has clarified for us the present known position of the endocrines in obstetrics and gynæcology.[1] Some chapters are fascinating in their interest; others, such as that dealing with embryology, are very condensed and need intense concentration. All, however, are well written and instructive.

There is a large bibliography at the end of each chapter, representing an enormous amount of reading in world literature. Very occasionally nomenclature is a little confusing, and we do not approve of the inclusion of an occasional modern American slang word in scientific literature.

Animal experiments form the bulk of the experimental work, and naturally the methods and results are not always applicable to man. Consequently many of the conclusions drawn must remain theoretical in regard to man. But from these animal experiments certain lines of treatment have been suggested and tried, and if in many of the cases, for example in primary amenorrhœa, the results are somewhat disappointing, at least they are beginnings from which valuable lessons can be learned.

The interrelation of the hormones and the possible existence of a sex centre in the mid-brain form an interesting chapter, but the experimental evidence is conflicting to a great degree, and naturally inconclusive. Kurzrok places the ovulation period soon after the fourteenth day, in which he agrees with the majority of workers, but does not believe in a "safe period". He contradicts the well-known findings of Knaus, and states that the uterus reacts to pituitrin in all phases of the menstrual cycle, and supports his statements with experimental evidence.

A glimpse is given of the immense possibilities of organic chemistry in the production of hormones, and when hormones can be synthetically produced their therapeutic value will be greatly increased.

The case histories of secondary amenorrhœa and intersexuality are most interesting and illustrate the wonderful opportunities that American workers have for investigating these hormonal dysfunctions. Kurzrok is still doubtful whether any new primordial follicles occur after birth.

Every medical practitioner will find this book useful and it will give him a clear conception of our present knowledge of hormones in regard to obstetrics and gynæcology. This subject, however, is still being actively investigated, and theories which are firmly held today may be exploded or proved as facts tomorrow.

[1] "Developmental Abnormalities of the Eye", by I. Mann, D.Sc., M.B., B.S., F.R.C.S., with a foreword by Sir J. H. Parsons, C.B.E., D.Sc., LL.D., F.R.C.S., F.R.S.; 1937. London: The Cambridge University Press (for the British Journal of Ophthalmology); Australia: S. Jaboor. Royal 8vo, pp. 453, with illustrations, of which many are in colour. Price: 50s. net.

[2] "Emanotherapy", by F. H. Humphris, M.D., F.R.C.P., D.M.R.E., and L. Williams, M.D.; 1937. London: Baillière, Tindall and Cox. Demy 8vo, pp. 198, with illustrations. Price: 7s. 6d. net.

[1] "The Endocrines in Obstetrics and Gynæcology", by R. Kurzrok, Ph.D., M.D.; 1937. London: Baillière, Tindall and Cox. Medium 8vo, pp. 504, with illustrations. Price: 34s. net.

The Medical Journal of Australia

SATURDAY, APRIL 2, 1938.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

CHILD DISCIPLINE.

In a recent number of *The Lancet*, R. Miller[1] takes child discipline out of the hands of the more advanced present-day psychologists and places it in its correct category as a medical problem; and yet, having done so, he concludes by confessing that it is by no means clear to him why a medical training should be supposed to impart any special insight into the ways of normal children or their management. It is curious that Miller should have failed to see that this very insight into the early training of the child is the great prophylactic measure against the psychoneuroses of the adult. W. H. R. Rivers, in his "Instinct and the Unconscious" (1920) bemoaned the fact that, while the medical profession had fully equipped itself for the medical and surgical problems of the Great War, it found itself with no such common body of principles and measures to deal with the psychoneuroses. And today the profession is ill-equipped to deal with the same problems in civil life. R. S. Ellery, writing in this journal recently, would seem from his investigations on psychiatry in the Soviet Union to have been convinced that the Soviet is right when, in order to build up adult efficiency, it has taken over the mental and physical training of the child. This training is regarded in Russia as a problem in preventive medicine — as a prophylactic measure against the psychoneuroses. The difference in outlook in Russia is well summed up in Ellery's statement that "England spends ten million pounds a year taking care of its mentally afflicted and less than ten thousand pounds on research into the cause of such afflictions".

The principles of child discipline cannot, however, be usefully discussed until the object of instilling discipline and the very meaning of discipline itself are defined. To many the word discipline conveys a picture of a very noisy and often inefficient sergeant-major—thereby symbolizing all those evils to childhood derived from ignorance and inefficiency—imposing his will on those whom chance has put under his control. Discipline, however, is defined in the army manuals as that process in training which leads the soldier to withstand the enemy's fire and to attain his objective. The word training implies an educable cortex and the possession of free will, for otherwise there can be no initiative. If punishment is necessary, as Miller states, there must be no feeling of injustice, and, further, after punishment the punished must be reinstated and the offence wiped out.

The principles of child discipline differ in no way from those of the army. The object of discipline is to fit the child to take its place efficiently in society with full possession of its free will. The object of discipline is therefore directed to the child and not to the appeasement of unreasonable and irascible old gentlemen, unless such appeasement enhances the social standing of the child. Neither is the object of discipline to satisfy any ill-conceived experiments of psychologists, which statement is not to be taken as a denial of the necessity for careful study of the principles of teaching on the part of all those responsible for child management. And it is here that the training of the medical pro-

[1] *The Lancet*, December 25, 1937.

fession in the first principles of neurology and psychiatry is required if the psychoneuroses are to be recognized in their earliest stages.

The question of child discipline is intimately bound up with that of school education; and surely an educated medical profession cannot indefinitely stand aloof in seeing punishments inflicted without reinstatement and without the offence being wiped out. And yet is it not a fact that failure, in such examinations of childhood as the school leaving and other examinations, inflicts punishment on pupils and parents without there being any provision for reinstatement or the wiping out of the offence? The investigation of the psychoneurotic effects of such failures should make it clear why medical training should give some special insight into the ways of normal, healthy children and their management. The duty devolves upon those in charge of medical education to see to it that medical graduates are suitably equipped to undertake this type of instruction.

Current Comment.

THE MECHANISM INVOLVED IN ARTERIAL HYPERTENSION.

The relation of vascular spasm to arterial hypertension might reasonably be described as a subject already well known, for it is believed that there is a more or less general contraction of the arteriolar bed in hypertensive states. But when proof is sought it must be admitted that this belief is not based on very positive evidence. Enid T. Oppenheimer and Myron Prinzmetal, in prefacing an account of their work on the rôle of the arteries in the production of hypertension, point out that it is probable on physiological grounds that the arterioles are the most likely portion of the vascular tree to be concerned, though narrowing might occur in any of its four subdivisions, namely, arteries, arterioles, capillaries or veins.[1]

These authors have presented some proof that it is indeed the arterioles that are the subject of a functional spasm. In summing up the known facts about hypertension, they state that survey of the literature seems to eliminate various hypotheses that have been put forward from time to time. Thus the underlying basis of hypertension is not a substantial increase in the cardiac output, nor an increased volume of blood, nor a raised viscosity, nor a purely localized spasm, such as in the splanchnic area; and it is not necessary to assume a constant structural narrowing, since relaxation of the hypertensive vessels is known to occur. Leaving out of the question the ultimate cause, whether neural or chemical or both, it remains to establish proof as to the site of what is apparently a very generalized vaso-constriction. Spasm of the retinal vessels has been observed in both primary and secondary types of hypertension and the radial artery has also been shown to be in a state of increased muscular tone in nephritis. But Weiss and others found that the arteriolar pressure was increased in hypertension, though the results of their attempt to demonstrate radiologically a narrowing of the arteries through structural changes in the walls is not convincing. As Oppenheimer and Prinzmetal point out, it is extremely unlikely on mathematical grounds that such a proof can be made, even though structural changes exist, since so minute a change would produce so definite a rise in pressure. It may be remarked here that this very fact makes it all the more difficult to be satisfied that slight structural changes are not present, and indeed such a view is consonant with pathological experience. But the present authors, having thus logically narrowed their field, now proceed to describe their own observations. They used a Gärtner capsule, which is a small hollow metal cuff with a thin rubber lining which can be inflated and attached to an ordinary sphygmomanometer. This was placed round a finger and observations were made of the pressure required to obliterate the digital artery. Comparison of the digital arterial pressure is thus easily made with the brachial pressure, and the pressure gradient from medium-sized artery to small artery. The subjects studied were fifty-one patients with raised blood pressure, fourteen with low blood pressure, and twenty-five normal controls. The hypertensive patients included persons illustrating both the benign and malignant types, as well as a number illustrating the secondary hypertension of nephritis, and one patient suffered from a primary tumour of the suprarenal gland. In ordinary hypertension it was found that there was no increase in the pressure gradient from brachial to digital artery. In other words, the relative conditions of arterial muscular tone in these vessels preserve their usual relations, and the increased resistance must occur in vessels smaller than the digital arteries. In contrast with this result, it was found in patients with obliterative vascular disease that the pressure gradient was increased owing to structural changes; and as a matter of interest the patient with an adrenal tumour also showed a great increase owing to constriction of arteries larger than the digital artery, through excess of circulating adrenaline.

The work of Oppenheimer and Prinzmetal, then, indicates that there is no increased resistance in arteries larger than the digital artery in the common primary and secondary forms of hyper-

[1] *Archives of Internal Medicine*, November, 1937.

tension. It is interesting to have more definite information based on experimental work, for at the present time, as pointed out previously in these pages, hypertension is a subject of great current interest, owing in part to our growing knowledge concerning the sympathetic nervous system and in part also to the work of modern neurosurgeons on operations designed to relieve various conditions due to vascular spasm. It is also interesting to look back and to see how the general introduction of sphygmomanometry into clinical medicine raised a wave of enthusiasm for the use of sundry vaso-dilator drugs; how these drugs were in the main found wanting and discarded; how organ extracts were tried and met with faint support; how the spastic element in hypertension was then regarded as an unfortunate physical factor in disease largely beyond our control; how a more logical attempt to deal with it was then attempted through lessening by drugs, such as the barbiturates, and also by psychological methods the assault of life's strains on the interbrain; and finally how by drastic surgical procedures attempts were made to reduce the constant though variable vaso-constriction. The mechanism seems now established; the causes are still obscure; but the means of relief, though yet wanting, would seem to be discoverable in the future.

THE CAUSE OF MONGOLISM.

Among the many possible forms of bodily and mental defect that may occur in children, the so-called mongolian type of mental deficiency occupies a place of its own. The children are generally so cheerful and tractable that the degree of their deficiency is even more reluctantly recognized than usual by the parents; the facial appearance is striking and, while disconcerting, not repulsive; and, lastly, the cause is obscure and the subject of much disagreement. Adrien Bleyer points out that advanced maternal age has received considerable attention as a cause, though authorities cannot agree as to its importance.[1] Bleyer has collected data from many sources, chiefly American, concerning 2,800 cases of mongolian idiocy. Such an inquiry is naturally a purely statistical one, and as many possible causes were considered, detailed data were necessary. Some of the previously suggested causes were soon dismissed, such as advanced paternal age and ultimogeniture *per se*. Primogeniture and immaturity of the mother cannot be dismissed so easily, for it has been stated that if the offspring of a first or second pregnancy is a mongol the mother is usually young. Bleyer's figures do not support this conclusion, however, and he agrees with those writers who place advanced maternal age as the single most important factor. He found the peak maternal age in the general population of America to be twenty-four, whereas the peak age of the mothers producing mongoloid infants was forty-one. Reproductive exhaustion, however, did not seem to be relevant, and the figures seem to show that the exhaustion which might be expected as the fecund period draws to a close of itself does not increase the liability of a mongol to be born, even when due correction is made for the smaller numbers of children born as members of larger families. So, too, primiparity of itself did not appear to be important. An allied factor is that of a long interval preceding the birth of a child. Quite a large number of authorities consider this as of great importance, and as distinguished an authority as Still suggested that when the possibility of any defect in offspring was thought of, an interval of six years since the birth of the last baby might be considered long, but an interval of twelve years dangerous. Bleyer is, however, inclined to agree with Penrose that the long interval is not of causal significance, though he considers the data as yet insufficient for accurate judgement.

Nobody could read the opinions of the writers who have made such earnest inquiries into this subject without thinking that, whatever is the ultimate cause of mongolism, a relatively advanced maternal age must be regarded as a factor. The only moral to be drawn is that it is better on these grounds (as on many others) for women to bear children early in life than late.

[1] *The American Journal of Diseases of Children*, January, 1938.

POST-GRADUATE LECTURES IN MELBOURNE.

The Melbourne Permanent Post-Graduate Committee has arranged an attractive programme for 1938. The chief feature will be a series of lectures by Dr. W. B. Castle, of the Thorndyke Memorial Laboratories, Boston, United States of America. Dr. Castle is best known by reason of his work on the rôle of *achylia gastrica* and of extrinsic and intrinsic factors in the ætiology of pernicious anæmia. A member of the staff of the Boston City Hospital, W. B. Castle enjoys a high reputation as a clinician and a teacher. He will give a series of lectures in Melbourne, six in number, from August 15 to 26, 1938; they will deal with anæmias, hæmorrhagic diseases and vitamin deficiencies. The fee for the course will be three guineas. From September 5 to 17, 1938, a course in obstetrics and gynæcology will be held at the Women's Hospital. The general refresher course will be held from August 15 to 27 inclusive. The fee for each of these two courses will be three guineas. One of the most satisfactory of the activities of the committee is that it provides special facilities for those desirous of undertaking clinical work in special subjects or in general medicine and surgery. Personal contact with members of public hospital staffs is arranged. A registration fee of one guinea is charged. A booklet containing details of the activities of the committee may be obtained from the honorary secretary, 33, Collins Street, Melbourne.

Abstracts from Current Medical Literature.

RADIOLOGY.

The Radiological Diagnosis of Intraabdominal Hernia.

Fay K. Alexander (*American Journal of Roentgenology*, July, 1937) reports five cases of apparent intra-abdominal hernia. The radiological diagnosis of intraabdominal hernia is made primarily by Röntgenoscopic observation at short intervals as the barium meal passes through the small intestine. The usual radiological examination of the stomach and duodenal cap will not enable one to diagnose this condition or to justify the statement as to its absence. In the patients in whom a diagnosis was made, the distal duodenum and duodeno-jejunal junction were situated abnormally to the extent that the opaque meal did not follow the usual anatomical course. It is not believed, however, that a variation in the anatomical course of the duodenum and duodeno-jejunal junction is in itself necessarily an indication of intraabdominal hernia, but in that this observation was found in every instance reported, it should be a warning of such a possibility. The small intestine may occupy the right side, mid-portion or left side of the abdomen. The coils of intestine are grouped very closely. There may be considerable churning, regurgitation and even distension of the small intestine with the presence of fluid levels, and it may be possible to demonstrate the point at which the small intestine enters the hernia. The loops of bowel present an appearance and configuration as though they are contained in a sac or confining boundary, rather than that they are being allowed the freedom of the abdomen justified by the length of the small intestinal mesentery. There is little movement on manual palpation, and a change in position or posture of the patient produces little, if any, change in the relation of small intestine to the abdominal cavity. Manual pressure on one area of the mass of gut is transmitted throughout the mass in a manner similar to the transmitted fluid waves in ascites.

Bronchiectasis.

F. P. Lee Zander and M. Davidson (*British Journal of Radiology*, February, 1938) state that massive collapse of the lung is a necessary and invariable antecedent to bronchial dilatation. The traditional view that infection is primarily responsible for the production of bronchiectasis is no longer tenable. In the absence of infection bronchiectasis *per se* is a benign condition. A large plug of mucus may completely block a main bronchus. Air distal to this block is absorbed and a sudden suction action is produced, causing approximation of the bronchi and retraction of the plug, which is drawn partly down into the lower lobe bronchus and partly into the upper lobe bronchus. This is further aided by inspiratory efforts. The direction of the upper lobe bronchus is altered and the bronchus is dragged down. (If the plug is large, then the bronchus to the upper lobe will be obstructed as well and a complete massive collapse will occur.) With further retraction of the plug the mouths of various bronchi are encountered and the plug gets split into smaller pieces. Occasionally a small bronchus may escape plugging. The bronchi that escape plugging lead to air-containing alveoli, and consequently the increased tug will be felt on these alveoli and they will become emphysematous. The depth to which the plug is retracted will vary in different individuals, according to the degree of aeration at the moment of complete blocking. In some instances the plug will come to rest in the finest bronchioles (at the *ductus alveolaris* or even in the atria), and these cases will show saccular dilatations. In others complete absorption of air will occur when the plugs have reached small bronchi, and these will show fusiform and cylindrical dilatations. Many of these collapsed lobes may originate from intrauterine life, and then are due to the presence of amniotic fluid in the bronchi at the time when the first inspiration occurs.

The Action of X Rays or Radium on Inflammatory Processes.

Arthur Desjardins (*Radiology*, October, 1937) discusses the X ray treatment of various inflammatory processes. Having treated some hundreds of patients, he states that although in some cases radiotherapy has not had a favourable effect, an unfavourable action has never been observed. In circumscribed inflammations the significant rôle of lymphocytes, polymorphonuclear cells and eosinophile cells in the defence of the organism against infection and the sensitiveness of these cells to irradiation make it appear likely that, when an inflammatory process is irradiated, the rays act mainly by destroying a proportion of the leucocytes infiltrating the lesion or circulating in the blood vessels which supply the infected area. This view is corroborated by the rapidity with which the symptoms often abate, and the physical signs disappear. Moreover, microscopic examination of irradiated inflammatory lesions has repeatedly shown destruction of leucocytes, especially lymphocytes, to be the most prominent feature observed. It seems logical to conclude, therefore, that destruction of leucocytes is the primary and direct effect of irradiation. As a result of the disintegration of infiltrating leucocytes the antibodies, ferments and other protective substances which these cells contained are liberated in the surrounding tissue spaces, where they become mixed with the tissue fluids. It is also probable, as the experimental evidence indicates, that the next step is an increase in phagocytosis by reticular cells which become macrophages. No doubt other intimate secondary or indirect effects related to cell metabolism are produced, but the precise character and significance of these effects are not clear.

Tumours of the Jaw.

H. M. Worth (*British Journal of Radiology*, March, 1937) discusses the radiographic appearance of odontomes—tumours arising only in the jaws because of the presence there of dental tissues. Epithelial odontomes arise from the dental epithelium alone, and include dental cysts, dentigerous cysts and multilocular cysts (adamantinoma). Composite odontomes arise from the dental epithelium and dental mesoblastic tissue, and comprise the complex, compound, geminated and dilated types. Dental cysts are by far the commonest tumours found in the jaws. They occur at all ages and with equal frequency in the mandible and maxilla. They are derived from epithelial cells, which are normally present in the periodontal membrane and which proliferate as a result of irritation from an infected tooth. A granuloma is formed, which eventually shows liquefaction of the central cells. The peripheral cells continue to multiply and break down, and a dental cyst results. The radiographic features are quite typical—an area of bone destruction having no structure in the centre, but with a clear and usually well-defined cortex. There may be one or more apices in the cyst cavity, or there may be no teeth or roots present at all. Some care is necessary in attempting to estimate the number of teeth which are involved in any particular cyst, because the cyst may be superimposed over but not involve many teeth. The destruction of the *lamina dura* will indicate which teeth are involved. Usually there is no difficulty in establishing the diagnosis of a dental cyst on radiographic evidence, but in those cases in which the cyst is closely related to the antrum there may be the utmost difficulty, and occasionally it will be found impossible to differentiate a cyst from the normal antrum. Dentigerous cysts are much less common and usually occur in young subjects. The radiographic appearances are quite typical and resemble those of a dental cyst, but with some part of a tooth or teeth situated in the cavity. The cortex is clearly defined, if not always so definitely as in the case of dental cysts. Multilocular cysts or adamantinomata are relatively rare and occur almost

entirely in the mandible, usually in the posterior portion. They probably arise from the epithelium of the dental organ, but this is by no means clearly established. Histologically there are two types, the "solid" and the "cystic", but the latter may reveal two rather distinct radiological appearances. Complex composite odontomes are irregular masses of enamel, dentine and cementum, and vary very considerably in size. They have a fibrous capsule, and may occur in any part of the jaws. In a radiograph a complex odontome appears as a very dense opacity having no regular structure or shape. It is separated from the surrounding bone by a narrow dark space, which is the site of the fibrous capsule. In the molar region a complete tooth may be present beneath the odontome. The great density and the presence of the capsule differentiate it from an osteoma. Compound composite odontomes may be non-cystic or cystic. The former variety resembles the complex one, but instead of being one solid mass it is made up of any number of separate pieces, and it is the recognition of this which enables the diagnosis to be made radiographically. Sometimes the individual masses resemble small badly-formed teeth or denticles, and have separate fibrous capsules. The cystic type is less common and resembles a dental or dentigerous cyst, but differs from them in that there are denticles or pieces of tooth substance contained in the cyst. Geminated odontomes is the name given to fused teeth or denticles unlike those of the normal series. By gemination is meant the fusion of two or more teeth by one or more dental tissues.

PHYSICAL THERAPY.

Carcinoma of the Larynx.

DOUGLAS QUICK (*American Journal of Roentgenology*, December, 1937) has endeavoured to make a sharp distinction between the clinical problems attendant upon the treatment of carcinoma of the larynx and those pertaining to the treatment of cancer of the hypopharynx. He is convinced that our foremost aid at present in the treatment of laryngeal cancer is X radiation applied after the basic principles of Coutard. He reviews briefly the surgical experience during the past century, and the development of radium treatment for this disease. In his opinion radium now occupies a more limited and more definite position in the laryngeal cancer field. Total laryngectomy has been rendered obsolete by the present status of radiotherapy, and the more conservative operation of thyreotomy for access, and at times for drainage, is extremely valuable in conjunction with irradiation, particularly with the use of gold radon seeds. Early laryngeal cancer is as favourable for irradiation as for surgical extirpation and the ultimate result is far better. The real problem is the more advanced case, in which surgeons never have been interested. The value of local and constitutional care of the patient during deep X ray therapy is emphasized, and the need for much further work, both clinical and pathological as well as physical, to obtain the ideal technical factors in this treatment of the disease is urged.

Review of Literature on Experimental X Ray Therapy.

W. F. MANGES AND R. M. SMITH (*American Journal of Roentgenology*, November, 1937) have endeavoured to find from the literature on research work the manner in which X rays benefit infections and inflammations. They regard the conclusions of Mischtschenko and his co-workers as offering the best explanation. These conclusions are briefly: (i) The optimum dose for inflammatory foci is 20% to 30% skin erythema dose for humans. The beginning stage requires higher dosages, whereas the healing stage requires weak dosages, intensive radiation during the regenerative stage being only detrimental. (ii) Irradiation produces an increase in destruction of leucocytes, an increase of phagocytosis and of the histiocytic reaction. These correspond to the clinical course whereby the inflammatory focus becomes smaller and the recovery period considerably shortened. Very high doses cause a break in the defence barriers and spread of inflammation. (iii) Irradiation calls forth non-specific antibodies in the blood. (iv) Röntgen rays influence colloids, so that there is a lowering of osmotic pressure, a lessening of exudation of wandering cells, and a conversion of surface tension. (v) Amino-acids and trypsin in inflammatory foci are increased, and there is also an increase in total protein. (vi) There was no evidence of direct irradiation effects on the blood vessels in experiments on the rabbit's ear. (vii) The relief of pain results from the action of stored protein split products and lipoids on the nerve endings, the lessening of tension and œdema, the transforming of tissue juices obtained from colloids and solution of the crystalloids.

The Production of Supervoltage X Rays by Means of an Electrostatic Generator.

R. DRESSER, J. TRUMP AND R. VAN DE GRAAFF (*American Journal of Roentgenology*, November, 1937) describe a new type of supervoltage generator for the production of short wave X rays, which has been developed at the Massachusetts Institute of Technology. The high voltage source is a direct current electrostatic belt generator which operates normally at one million volts, and at this potential can deliver to the target of the tube a current of three milliampères. The X ray tube is of the cascade type, and is continuously evacuated by a high speed mercury vapour pumping system. Because of the constant potential, a homogeneous beam of high speed electrons is produced, which results in an exceedingly high Röntgen output and short effective wave length. At a potential slightly higher than one million volts, with a focal skin distance of 70 centimetres, a filter equivalent of six millimetres of lead, and current of one milliampère the output is 60 *r* per minute. The control of the apparatus is simple, and the usual trained technician can learn to operate it in a few minutes. The total power input required to run the unit is small, being only about fifteen kilowatts.

The Place of X Rays in the Treatment of Certain Forms of Chronic Arthritis.

F. HERNAMAN-JOHNSON (*British Journal of Radiology*, November, 1937) gives a description, both clinical and radiological, of those forms of arthritis in which X rays should be given a trial. He states that the local application of X rays is of great value in cases of hypertrophic non-infective osteoarthritis; it is worth trying in chronic infective arthritis, and may give local and temporary relief in rheumatoid conditions. It is valueless in true degenerative arthritis in the very old. The author is also of the opinion that the constitutional use of X rays is of great value in checking the otherwise inevitable advance of spinal arthritis in young people, and its possibilities in the treatment of rheumatoid arthritis are worth investigating. In constitutional treatment by X rays the whole trunk must be treated, the voltage should be comparatively low, and the greatest care must be taken not to give an overdose.

Treatment of Epitheliomata of the Naso-Labial Fold.

O. N. MELAND (*American Journal of Roentgenology*, November, 1937) states that carcinoma in the region of the naso-labial fold has a characteristic line of spread, namely, along the lines of embryonic fusion, which represent the lines of least resistance. He advocates protracted fractionated radium therapy as the method of choice; but to be successful care should be taken to give treatment over a fairly wide area, and to make sure that the depth dose is sufficient. Protracted X ray therapy will give results similar to those of radium therapy. In recurrent lesions the possibility of success is dependent upon the presence or absence of necrosis of cartilage or bone which is secondarily infected. When adequate irradiation is not successful, electrocoagulation followed by plastic reconstruction at a later date is advocated.

British Medical Association News.

SCIENTIFIC.

A meeting of the Victorian Branch of the British Medical Association was held at the Medical Society Hall, Albert Street, East Melbourne, on February 2, 1938, Dr. F. L. Davies, Senior Vice-President, in the chair.

Prostatic Surgery.

Dr. G. R. Davidson read a paper entitled "Some Experiences of Prostatic Surgery in a Provincial City" (see page 611).

Dr. J. Thomson Tait opened the discussion. He congratulated Dr. Davidson on his paper, particularly as it had been a study based on personal experience and characterized by straightforward statements. Dr. Tait recalled that the late Mr. Hamilton Russell had stated that he would rather hear of the clinical experiences of a lecturer than a dissertation on theories and cullings from literature. Dr. Tait said that he had often been struck by the alarming statements about the mortality of prostatectomy made to him by members of the lay public. The mortality rate was firmly believed to be in the neighbourhood of 25%. Such statements, it was true, might have been read in medical journals, but Dr. Tait was convinced that the mortality rate should not be anywhere near 25% and that a rate of 5%, as in Dr. Davidson's series, should be much nearer the mark. Another statement that was commonly made to Dr. Tait by patients or their friends was that if the patient survived the operation he was likely to be crippled afterwards. In particular, reference was made to fistulæ and to persistence of sepsis. He believed that over-statement of the position had created false impressions, and that the complications were less frequent and less formidable than was generally supposed. He considered that the mortality rate of 5% should be obtained, except for elderly men, who looked on prostatism as the last stage of a long life, wanted only relief from pain, and regarded the operation as an easy way out of a miserable existence.

Dr. Tait then referred to the indications for operation, and gave expression to what he regarded as the "golden rule": the operation should not be performed on a patient who had frequency only; obstruction was the indication for operation. The establishment of the presence of residual urine to the extent of approximately four ounces was a good general guide to the necessity for operation, provided that the age of the patient was taken into account. In the older group that amount of residual urine was relatively unimportant, and the patient might be left to see his life out without operation. In the younger group of patients under sixty years of age it might be considered desirable to operate if the patient had only two or three ounces of residual urine.

Speaking of prostatic resection, Dr. Tait said that he was glad that Dr. Davidson had emphasized that this procedure was not easy and not as simple and innocuous as had been indicated by certain people in writing and in speaking. The indications for resection were the same as those for prostatectomy, and the sole criterion of success was an empty bladder subsequent to the operation. Dr. Tait qualified this statement by referring to the necessity for the size of the urethra to be large enough to allow of the passage of the big instrument which had to be used. The urethra could be dilated progressively by indwelling catheters appropriately graduated in size. On occasions he had successfully allowed of the passage of the instrument by means of a preliminary internal urethrotomy. Bleeding from the prostate and the dangerous risk of sepsis were complications of resection which were serious and difficult to avoid and to overcome. The pre-operative preparation before resection needed to be performed just as carefully as for prostatectomy. It had to be recognized, too, that the operative trauma of resection took almost as long to heal as that of prostatectomy. He concurred in Dr. Davidson's statement with reference to the acutely infected cases. It had been his experience that recent infection was fraught with more danger than was chronic infection.

At this stage Dr. Tait drew diagrams to illustrate the points of entrance of sepsis and the most likely sites for troublesome hæmorrhage, and demonstrated that it was only safe to resect the portion between the verumontanum and the internal sphincteric bar. He considered that for successful resection patients had to be carefully selected. The ideal type of prostate for resection was the one which presented a simple crescentic posterior enlargement. The main body of the crescent could be resected, but he thought it was safer to leave the two corners, where there was extra risk of bleeding. When, in addition to the simple crescentic appearance, two lateral lobes were enlarged, the lateral lobes were liable to fall together after resection of the trigonal portion and would then block the urethra. It was thus necessary to warn patients that it might not be found possible to complete the resection at one sitting. In conclusion, Dr. Tait again congratulated Dr. Davidson on his results, which were excellent and showed that a low mortality rate was attainable.

Dr. Cedric Roche spoke in support of resection. He said that during his recent visit to the United States of America he had been very impressed, particularly with the work of Gershom Thompson at the Mayo Clinic, who had practised resection extensively and had reduced the mortality rate to 0·7%. He had performed resection for prostatic obstruction in as many as 250 consecutive cases without a death. Thompson did not pick his patients unfairly; he included those with blood urea up to 100 milligrammes per 100 cubic centimetres of blood after he had failed to reduce the blood urea level by other means, and he included many examples of infected bladder due to trabeculation and diverticulæ. Dr. Roche was convinced that in the hands of an expert such as Thompson these splendid results could be achieved. Dr. Roche had treated over sixty patients by resection. He had not been able to carry out the procedure on patients with big bulging prostates because of mechanical difficulties and because the bleeding proved too baffling; but the usual prostatic enlargements up to a medium size as well as the fibrous prostates and bar formations had been dealt with. In the series he had lost four patients. Two of these were patients in whom he should not have attempted the procedure because of too advanced malignant disease. The other two patients who had died were very old men with heart disease who had refused to have a suprapubic catheter. Hæmorrhage had not been a particular worry in the series, but infection was the difficulty. The after-treatment of the bladder had to be conducted very carefully and he had been fortunate to have capable colleagues on the spot to attend to that for him. He affirmed that the future hope of patients with enlarged prostates lay in resection rather than in prostatectomy. He could achieve better results by resection than by the suprapubic route.

Dr. Julian Smith, junior, said that he had to speak in defence of the work of the surgeons at the Royal Melbourne Hospital. Dr. Davidson had referred to the deaths after prostatic surgery at that hospital over a certain period. Dr. Julian Smith had been through the records for the past twelve years and had found that 810 patients had been admitted on account of non-malignant enlargement of the prostate gland; of these, 550 had been subjected to prostatectomy and 103 had died; the mortality rate was approximately 18·5%. The Royal Melbourne Hospital surgeons would envy Dr. Davidson, who appeared to be working in the Utopian field where patients with acute retention could be admitted to hospital. In the Royal Melbourne Hospital series to which Dr. Julian Smith had referred, 560 of the patients had presented acute retention of urine. He had included under that heading patients in whom it was noted in the histories that the residual urine amounted to over thirty ounces or that the bladder was enlarged up to the level of the umbilicus. The bladders of those patients had been catheterized

repeatedly in the casualty department, and many of them had been admitted to hospital because of the probability of approaching death. For that reason it was not right to lay the mortality rate at the door of suprapubic prostatectomy. In contrast, and with reference to transurethral resection, Dr. Julian Smith was not prepared to accept without question "the moonlight and roses" from the United States of America. He had read of the prowess of a surgeon who had operated on 950 patients by transurethral resection in three years. The surgeon had not been in a centre such as New York or Chicago, with a very large population, but had worked in a city with a population approximately twice that of Melbourne. If in twelve years only 810 patients had been admitted to the Royal Melbourne Hospital with non-malignant enlargement of the prostate gland, and if this single operator had been able to perform transurethral resection on 950 patients in three years, Dr. Julian Smith was content to state those facts and to allow people to draw their own conclusions. The operation would forever be in disrepute if it was performed for vesical irritability in the absence of obstruction.

Dr. Davidson, in reply, expressed his appreciation of what had been said by the speakers who had followed him. He hastened to assure Dr. Smith that he had not intended to reflect in any way on the skill of the surgeons at the Royal Melbourne Hospital, but to emphasize the importance of earlier admission to hospital and earlier operation. He was glad to have heard from Dr. Tait of the method of using a series of indwelling catheters for dilating the urethra when necessary for the passage of the instrument for resection. He thought that Dr. Roche would agree that the difficulties encountered by a man doing his first fifty or one hundred resections would be very great in comparison with those of a man who had had much experience. Dr. Davidson illustrated the need for watchful and efficient after-care by giving in detail an account of an occurrence which he had experienced recently. In a period of not more than twenty or thirty minutes which had elapsed between two consecutive visits he had paid after operation to a patient, the patient's bladder had become full of blood clots and the pulse rate had risen to over 150 beats per minute. He had managed to break up and remove the clots, and had passed a Foley's bag and controlled the hæmorrhage by inflating the bag. In that case the patient had nearly died. The surgeon was very much at the mercy of a nurse on those occasions. When risks of that magnitude were possible, transurethral resection could not be termed a minor or safe procedure. Nevertheless, Dr. Davidson felt certain that it would occupy an important place in the prostatic surgery of the future.

DR. F. L. DAVIES, from the chair, commented on the relative infrequency of addresses by members from the country at the monthly meetings of the Branch. He said that Dr. Davidson had delivered a most helpful and stimulating paper, which had been greatly appreciated by those city members who were present at the meeting.

At the conclusion of the meeting, Dr. R. Wright-Smith demonstrated from a series of preparations from the pathological department of the Royal Melbourne Hospital. He produced a range of specimens to illustrate the various types of prostatic enlargements which presented the problems that had to be overcome by the surgeons. The demonstration included a number of photomicrographs obtained from sections of the specimens.

NOMINATIONS AND ELECTIONS.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Corlette, Ewan Laurie, M.B., B.S., 1931 (Univ. Sydney), 15, Wentworth Street, Point Piper.

Morson, Stuart MacKenzie, M.B., B.S., 1938 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Medical Societies.

VICTORIAN MEDICAL BENEVOLENT ASSOCIATION.

THE annual meeting of the Victorian Medical Benevolent Association was held at the British Medical Association Hall, Albert Street, East Melbourne, on March 4, 1938.

Election of Office-Bearers.

The election of officers for the year 1938 resulted as follows:

President: Dr. R. H. Fetherston.
Vice-Presidents: Dr. J. P. Major and Dr. D. Upjohn.
Honorary Secretaries: Dr. E. L. Gault and Dr. F. Newman.
Honorary Treasurer: Dr. L. Mitchell.
Committee: Dr. W. Newton, Dr. Fay Maclure, Dr. A. P. Derham and Dr. C. H. Dickson.

Financial Statement.

The audited balance sheet presented by the honorary treasurer, Dr. L. Mitchell, showed income £500, made up of subscriptions £173 and interest on investments £327. The expenditure was £430, leaving a balance of income over expenditure of £70. The statement of funds and investments showed a total of £8,907, of which £8,481 consituted the permanent fund, and the balance, £426, the current account.

Annual Report.

The report acknowledged the valued cooperation of the council and secretary of the British Medical Association in securing the subscriptions of a large proportion of members, and earnestly appealed for a continuance of that support. In view of the heavy calls upon its funds and the prospect of greater calls in the future, the committee commended its good work to the generosity of the profession. In addition to the annual subscription, larger donations either to the permanent fund or to the current account for relief of expenditure would be welcomed. The committee also desires to direct the attention of those who have made or are making their wills to the work of the association.

Correspondence.

PURPURA FULMINANS.

SIR: On June 21 last I was called to a child, aged eighteen months, a well-nourished male that had not previously needed any medical attention. The messenger, the child's father, told me that it had been slightly feverish for some hours, and for a couple of hours before he left home (two hours' journey distant) had suffered griping abdominal pain and had passed two motions, each containing a little bright blood. Praying that I might be wrong, but fearing that I might be called upon to treat a case of intussusception under conditions of extreme difficulty, I made hurried preparations and accompanied the father to his home on one of the islands of Franklin Sound. On arrival I was overjoyed to find that all symptoms had completely subsided. No rise of pulse rate or temperature, no tenderness, no rigidity and no sign of a lump in the abdomen were present; there was slight pallor only. The child made some display of appetite. I let well enough alone, with strict instructions to report any change instantly. I see the father every few weeks, and a week ago made the second inquiry since the original attendance and was told that they had thought once, about a week before, that another attack was threatened, but dopiness and feverishness had been so slight in extent and duration that calling me in had not even been considered.

On the morning of the ninth instant I received an urgent call to go to the same child. The message said: "Child was quite well at 3 p.m., playing actively with others, but as the day wore on began to get hot and drowsy. The mother took the temperature at 11 p.m. It was 105° F., but the child did not seem really ill and was certainly in no pain. It woke at 3.30 a.m., apparently much better, but at daylight it was very bad, with purple blotches appearing on the forehead and behind the ears." Of course I realized then what the threatened intussusception had been, and hurried away. But the child died at 8.30 a.m., before my arrival. I wrote a certificate of death from *purpura fulminans*. But as I have since noted that Sequiera (1919) states that there were (only) 65 recorded cases to date, I thought it best to record this case.

I regret that I did not examine the whole body carefully so as to give a full account of the extent of the ecchymoses—on the parts I saw there were more of them than of normal skin—nor did I bother to get an exact chronology of the different signs and symptoms.

The oldest child by a former marriage of the same father had a similarly sudden death at the age of two and a half years about twenty-five years ago, but I got no trustworthy account of the symptoms. If any further information is desired by any reader, I shall do my best to furnish it. My only excuse for vagueness is that I did not at the time of death realize the rarity of the condition and its consequent general interest.

Yours, etc.,

Theo. J. Ick.

Whitemark,
Flinders Island,
Tasmania.
August 12, 1937.

A PROTEST.

Sir: I would be pleased if you could afford me some of your space to voice a protest.

As a general practitioner I find it necessary frequently to refer patients to specialists for further advice and treatment; very often these patients for financial reasons are referred to a public hospital where they are treated by resident and honorary medical officers.

My first argument is with the young resident medical officer, who in nearly all cases has the diagnosis handed to him on a silver platter, and who, to impress the patient and relatives with his own knowledge, remarks something like this: "He should have been sent here a week ago; the doctor who has been treating him should not have done that" *et cetera*.

A case in point is a child whom I saw for the first time at the age of thirteen days. The mother complained that the child vomited after each meal and was losing weight. The child is a male and a first-born, and my first thought before seeing the child was congenital hypertrophic pyloric stenosis, and I examined the child on this occasion for fully half an hour, looking for visible peristalsis and feeling for a tumour. I found neither, and had been warned never to make the diagnosis without finding one or both. I saw the child again the next day and again the following day with similar results. In each case I examined the child for at least twenty minutes before and after a feed.

On the sixteenth day of the child's life, four days from the first visit, I definitely observed exaggerated gastric peristalsis and felt a tumour. I made the obvious diagnosis and advised operation.

At the mother's request I sent the child to a specialist, as she doubted my diagnosis. Apparently even he was not certain, for the child was X rayed, and on this the final diagnosis was based; and the child was operated on and a pyloric tumour was incised, with uneventful recovery.

To my amazement the patient's mother on her return said to me: "The doctor said the child should have been operated on a week ago, and the child nearly lost its life through the delay."

I maintain the diagnosis was made early, and on clinical symptoms and signs alone, and that the doctor who made the above remark was, if not unethical in his behaviour, at the very least uncharitable.

I number many Macquarie Street and Wickham Terrace specialists as good friends, and I hope the above instance is the exception rather than the rule.

I would, however, like to point out to those stern critics who rely for all or most of their diagnoses on the ancillary branches of the profession, and who depend on the general practitioner for their livelihood, that a general practitioner will be very cautious of sending another case to men who criticize their treatment and diagnosis to the patients or relatives.

I would be pleased to hear from fellow practitioners through your columns if they have had similar experiences.

I conclude this letter with a plea to "doctors and specialists" to be more charitable in their conversations in front of patients who have been sent to them by busy and overworked general practitioners.

Yours, etc.,

"G.P."

March 13, 1938.

HOW CIVILIZATION MANUFACTURES NEUROSES.

Sir: The article by Dr. John Bostock in your issue of March 5 should not only be read by every medical practitioner, but should form the base of thought and discussion.

I am afraid that I have not found the treatment of psycho-neurotic disorders as easy as Dr. Bostock, but I do not wish to comment further upon that aspect of this paper.

The idea underlying Dr. Bostock's remarks is that environment has a very big influence in the genesis of psycho-neurotic disorders, and that the present social order does not provide a suitable environment for large numbers of our population.

As I remarked in a previous letter, it is a very healthy sign to see members of our profession thinking and writing and speaking along these lines; far too long and much too intensively have we gazed into the magic wonders of the test tube and listened to the long details issuing from so-called laboratories for the solution of many of the problems of disease, when all that is required is to keep one's eyes open upon the social environment in which our citizens exist (one could not designate it "live"). The wonder is not that disease is so common, but that good health is so common. How can we hope to have health and happiness when so many of our citizens are born and live in a condition of poverty?

It is absurd to talk of the nation being prosperous when the vast majority of our breadwinners earn less than £3 per week; when unemployment still exists for many thousands. According to the latest Commonwealth Year Book, 70% of Australia's breadwinners get less than £3 a week.

Disease is a complex problem, but there can be no doubt that the totality of the positive and negative factors of poverty is one of the major factors in creating disease and perpetuating types that are born only to eke out an existence attending various hospitals.

It is plain that we cannot continue indefinitely as at present; somewhere, some time, the flood gates will break, and our present society, which has cost so much to build, will be broken perhaps irreparably.

The solution is really quite simple. It is not to be found in the hysterical waving of flags, the trumpeting forth of speeches of hate, five-year plans, organization of the masses for production, and all such psycho-neurotic manifestations, but in a little clear thinking; a return to man's condition of freedom, freedom from taxes on his industry, freedom from government interference, freedom from privileges, freedom of access to his common birthright, the earth. The fascist state, the socialist state, will

not breed men, only masters and slaves; to be a man one must be free, and to be a man means to have good health of mind and body.

Yours, etc.,
Paul G. Dane.

110, Collins Street,
Melbourne, C.1,
22nd March, 1938.

Obituary.

JOHN BERNARD GUNSON.

We are indebted to Dr. Bronte Smeaton for the following appreciation of the late Dr. John Bernard Gunson.

A good and upright man and a kind and capable doctor left us when John Gunson died at the age of sixty-nine. His father, a very early doctor in Adelaide (1856), was much loved and respected, and died a knight of the church. His piety and fastidious standard of duty and honour descended to his son and inspired his life. Gunson received his medical education at the University of Adelaide and in London. After terms as resident at the Adelaide and Children's Hospitals he conducted a large and important practice in Adelaide. He was a pioneer in the establishment of the first bacteriological and pathological laboratory in Adelaide at the Children's Hospital and in charge of the first X ray apparatus which was established at the Children's Hospital.

An honorary physician at the Children's Hospital, an honorary assistant physician at the Adelaide Hospital, and honorary consulting obstetrician of the Queen's home, he yet found time to give devoted and gratuitous service to the staff and inmates of many of the social and philanthropic institutions of his church.

He was in turn secretary of the South Australian Branch of the British Medical Association, secretary of the 1905 congress, vice-president of the Branch, and was for many years a valuable member of council. All had the greatest respect and affection for him, and his dignity and probity and gentle courtesy made him an example which did much to maintain and raise the standards of his profession. His services to the Saint John Ambulance Association gained the order of an Officer of Saint John of Jerusalem. A pioneer in motoring, he served as a foundation member of the Royal Automobile Association and remained a member of the council during his active life.

Despite failing health he worked on heroically until a series of interferences with his cerebral circulation brought him to a bed of suffering for two years. His son succeeds him in his profession.

PHILIP SYDNEY JONES.

We are indebted to Dr. F. P. Sandes for the following appreciation of the late Dr. Philip Sydney Jones.

When Sid Jones passed from amongst us the thoughts of many of his friends must have flown back to the days of thirty-five years ago, when there lived within a mile of the university a number of young medical men in strenuous competition with each other. This happy band of warriors helped each other with their work, and in their spare time were wont to amuse themselves with tennis, boat sailing, noisy motor cycles and cranky motor cars. Sid Jones, when an earnest and capable medical student, had represented his university in cricket, and in his early days of practice was an enthusiastic devotee of the game. But there came a new and insidious disease to which he and some of his fellows became victims, a game called golf, and it was interesting to see how stubbornly yet unsuccessfully he resisted the wiles of the "wee small ball". At the recent fortieth anniversary dinner of our old club he was so full of reminiscence of the old days that no one would have suspected him to be a pastmaster in the fascinating game and the head of a noted golfing family.

But he devoted no less industry to his work. The son, and yet in some of his attributes the opposite of Sir Philip Sydney Jones, one who had been an Australian pioneer in ovariotomy and lithotrity, and who subsequently became perhaps the leading analytic clinician of his time in New South Wales, a distinguished physician and public-spirited citizen, young Sydney Jones showed a clinical shrewdness and practical efficiency in his work that often surprised his medical friends. He concealed it under a free and easy manner and a cheery optimism, which with his ability endeared him to his patients and brought to him an extensive general practice. He had, too, a pretty wit, as shown in his reply to the young father who asked him about the fee for an impending christening: "If it is the same parson that married you, he ought to throw it in as after-treatment." After some years of hard professional work he may have suspected that he was laying the foundation of a "calcareous cerebral or a chalky coronary", so he went to Macquarie Street, pursued less strenuous practice, devoted a good deal of attention to anæsthesia, to business affairs and his favourite sport. He took his family abroad in search of fresh links and clubs that were new. And when it came to him that his days were numbered he faced it calmly and cheerfully, made no complaint, and went on with his game—a sportsman to the last. As he fainted on the fairway no doubt the last thoughts of the kindly husband and father were for the dear ones for whom he had worked and with whom he had played. And if there is a golfer's paradise in the Elysian fields, one can see old Sid chaffing the keeper of the gate, assuring him that he had had a pretty good time on earth, but thought it a bit hard that he had not been allowed to hole the last putt.

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

The undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1912 *and* 1915, of New South Wales, as duly qualified medical practitioners.

Conlon, Leonard Telford, M.B., B.S., 1938 (Univ. Sydney), 35, Enmore Road, Newtown.

Ellis, Archie Samuel, M.B., B.S., 1937 (Univ. Melbourne), s.s. *Duntroon*.

Harris, Joane Josephine Hughs, M.B., B.S., 1938 (Univ. Sydney), Saint Joseph's Hospital, Auburn.

Mackenzie, Kenneth MacIver, M.B., Ch.B., 1937 (Glasgow), c.o. Dr. Broome, Coonamble.

NOTICE.

Caricatures of Renowned Scientists of the Nineteenth Century, by the Famous Artist Spy, Available to the Medical Profession.

During the last century a London periodical, now out of print, caricatured world-famous men of medicine, science, law and politics. Petrolagar Laboratories and John Wyeth and Brother Incorporated have selected for reproduction a number of these studies interesting to members of the medical profession. Copies suitable for framing, together with a brief description of the subjects, will be sent to doctors upon request. The series is comprised of caricatures of such renowned scientists as M. and Madame Curie, Sir William Crookes, Thomas Henry Huxley, Sir Richard Owen, Charles Robert Darwin, Oliver Wendell Holmes, Louis Pasteur, Sir James Paget, Rudolph (Ludwig Karl) Virchow. Communications should be addressed to Petrolagar Laboratories Incorporated and John Wyeth and Brother Incorporated, 45, Brougham Street, Sydney.

Books Received.

A POCKET MEDICAL DICTIONARY, compiled by L. Oakes, S.R.N., D.N., assisted by T. B. Davie, B.A., M.D., M.R.C.P.; Third Edition; 1938. Edinburgh: E. and S. Livingstone. Crown 16mo, pp. 417, with illustrations. Price: 3s. net.

THE PRACTITIONER HANDBOOKS: PRACTICAL PROCEDURES, edited by Sir Humphry Rolleston, Bt., G.C.V.O., K.C.B., M.D., F.R.C.P., and A. A. Moncrieff, M.D., F.R.C.P., with a preface by Sir David Wilkie, O.B.E., M.D., M.Ch., F.R.C.S., F.A.C.S., F.R.S.E.; 1938. London: Eyre and Spottiswoode Limited, for The Practitioner. Demy 8vo, pp. 293, with illustrations. Price: 10s. 6d. net.

CHIROPODY THEORY AND PRACTICE, by F. Charlesworth, F.B.A.Ch., with a foreword by W. S. Creer, M.Ch., F.R.C.S.; Second Edition; 1938. London: The Actinic Press Limited. Demy 8vo, pp. 221, with illustrations. Price: 15s. net.

Diary for the Month.

Apr. 5.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Apr. 6.—Victorian Branch, B.M.A.: Branch.
Apr. 6.—Western Australian Branch, B.M.A.: Council.
Apr. 7.—South Australian Branch, B.M.A.: Council.
Apr. 8.—Queensland Branch, B.M.A.: Council.
Apr. 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Apr. 19.—New South Wales Branch, B.M.A.: Ethics Committee.
Apr. 20.—Western Australian Branch, B.M.A.: Branch.
Apr. 21.—New South Wales Branch, B.M.A.: Clinical Meeting.
Apr. 22.—Queensland Branch, B.M.A.: Council.
Apr. 27.—Victorian Branch, B.M.A.: Council.
Apr. 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Apr. 28.—South Australian Branch, B.M.A.: Branch.
Apr. 28.—New South Wales Branch, B.M.A.: Branch.
May 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.

Medical Appointments.

Dr. F. J. Ryan has been appointed Junior Medical Officer at the Parkside Mental Hospital, South Australia.

* * *

Dr. P. L. Grogan has been appointed a Certifying Medical Practitioner at Richmond, pursuant to the provisions of the *Workers' Compensation Acts* of Victoria.

* * *

Dr. S. Mathews has been appointed Medical Officer of Health to the Kalgoorlie Road Board, under the provisions of *The Health Act*, 1911 *to* 1937, of Western Australia.

* * *

Dr. W. J. Donald and Dr. A. J. Collins have been appointed Members of the Boards of Official Visitors to the Mental Hospitals at Gladesville and Callan Park, New South Wales.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xx and xxii.

Narembeen Road Board, Narembeen, Western Australia: Medical Officer.
Anti-Tuberculosis Association of New South Wales: Honorary Physicians.
Balmain and District Hospital, Balmain, New South Wales: Junior Resident Medical Officers.
The Rachel Forster Hospital for Women and Children, Sydney, New South Wales: Medical Officers.
Royal Melbourne Hospital, Melbourne, Victoria: Honorary Officers.
State Public Service Board, Brisbane, Queensland: Assistant Medical Superintendent.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
South Australian: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

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